

In the United States Court of Federal Claims
OFFICE OF SPECIAL MASTERS
No. 18-925V
(to be published)

JOSE GAMBOA-AVILA,

Petitioner,

v.

SECRETARY OF HEALTH
AND HUMAN SERVICES,

Respondent.

* Chief Special Master Corcoran

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* Dated: September 11, 2023

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Curtis R. Webb, Attorney at Law, Monmouth, OR, for Petitioner.

Colleen Clemons Hartley, U.S. Dep’t of Justice, Washington, DC, for Respondent.

ENTITLEMENT DECISION¹

On June 27, 2018, Jose Gamboa-Avila filed a petition for compensation under the National Vaccine and Injury Compensation Program (the “Vaccine Program”).² (ECF No. 1) (“Petition”). Petitioner alleges that a pneumococcal vaccine he received on November 13, 2017, caused him to incur Guillain-Barré syndrome (“GBS”). *Id.*

The parties have agreed that the matter could reasonably be resolved via ruling on the record and filed briefs in support of their respective positions. *See* Petitioner’s Motion, dated January 14, 2022 (ECF No. 72) (“Mot.”); Respondent’s Opposition, dated April 29, 2022 (ECF

¹ Under Vaccine Rule 18(b), each party has fourteen (14) days within which to request redaction “of any information furnished by that party: (1) that is a trade secret or commercial or financial in substance and is privileged or confidential; or (2) that includes medical files or similar files, the disclosure of which would constitute a clearly unwarranted invasion of privacy.” Vaccine Rule 18(b). Otherwise, the whole Decision will be available to the public in its present form. *Id.*

² The Vaccine Program comprises Part 2 of the National Childhood Vaccine Injury Act of 1986, Pub. L. No. 99-660, 100 Stat. 3758, codified as amended at 42 U.S.C. §§ 300aa-10 through 34 (2012) [hereinafter “Vaccine Act” or “the Act”]. Individual section references hereafter will be to Section 300aa of the Act (but will omit the statutory prefix).

No. 76) (“Opp.”); Petitioner’s Reply, dated June 2, 2022 (ECF No. 80) (“Reply”). In addition, counsel presented oral argument on February 13, 2023. ECF No. 83 (transcript of proceedings).

Having reviewed the above plus the filed medical records, expert reports, and associated literature, I hereby deny an entitlement award. As discussed in greater detail below, Petitioner has not preponderantly established that the pneumococcal vaccine can cause GBS—and this alone is a sufficient basis for dismissal. I have reached the same determination in several prior Program cases, based on a comparable theory, and nothing advanced by Petitioner *in this case* supports an alternative finding, or reflects new scientific/medical developments that would provide the occasion to reconsider my prior reaction to the theory.

I. Factual Background

The core facts of this case need not be given considerable time herein, since it is my determination that the claim’s resolution turns on the “can cause” causation prong.³ Petitioner, then 48 years old, received the pneumococcal vaccine during a routine visit for care of his existing HIV-AIDS diagnosis on November 13, 2017. Ex. 2 at 6–12. He then sought treatment on November 27, 2017, complaining of generalized body aches, a headache, night sweats, and numbness since November 13, 2017, the date of his last visit (although the accuracy of this record is disputed). *Id.* at 17–21.

The next day, Petitioner again obtained treatment, reporting ongoing complaints that petitioner reported had been present for seven days, including that his eyes felt swollen; his face and body felt numb; he was having difficulty eating and could not taste food; his legs hurt when he walked; he had difficulty sitting or standing for “too long;” and his hands were weak. Ex. 2 at 28–30. On examination, infectious disease specialist Leila Hojat, M.D., observed that Petitioner had a mild right-sided facial droop, with reduced sensation on the left side of his face and in both legs. *Id.* But he had full strength in all of his extremities. *Id.*

Petitioner went to the emergency room that very night, now complaining of whole-body muscle pain and numbness that he described as “tingly,” as well as facial weakness, and that these symptoms made it difficult for him to talk and walk. Ex. 2 at 31–38. Over the next several days, Petitioner was hospitalized for suspected GBS, and eventually diagnosed with it after discharge in December 2017. *Id.* at 63–64, 96, 192–93. There is, however, later evidence in the record in which physicians contested that diagnosis.

Treaters did discuss the vaccine and its potential relationship with Petitioner’s GBS beginning as early as November 28, 2017 and lasting until the end of the year. Such statements

³ The parties do disagree as to the accuracy of the GBS diagnosis (*see, e.g.*, Opp. at 13–18), and that this remains an open issue that would also require resolution if this Decision is reversed. But in the interests of brevity, I focus only on whether the pneumococcal vaccine can even cause GBS.

were discussed by ten treaters. *See, e.g.*, Ex. 2 at 30, 71–72, 76, 84, 90, 94, 96, 102, 105, 107, 112, 115, 120, 126, 130, 136, 141–42, 145, 151, 155, 207. His treaters discuss the temporal relationship of his vaccination to his GBS, with some discussing causality. *See, e.g.*, Ex. 2 at 30 (“[h]e was subsequently hospitalized and is suspected to have [GBS] from his recent Prevnar vaccine”), 72 (“[r]ecent vaccine may potentially predispose to GBS”), 76 (“[p]ossibly triggered by recent pneumonia vaccine”). However, other treaters simply repeat past impressions. *See, e.g.*, Ex. 2 at 87, 102, 105, 112, 155 (“[v]accine may have been trigger.”).

II. Expert Reports

A. Petitioner’s Expert – Lawrence Steinman, M.D.

Dr. Steinman, a neurologist, prepared four written reports for the Petitioner. Report, dated June 21, 2020, filed as Ex. 15 (ECF No. 38-1) (“Steinman First Rep.”); Report, dated April 25, 2021, filed as Ex. 39 (ECF No. 55-1) (“Steinman Second Rep.”); Report, dated September 5, 2021, filed as Ex. 46 (ECF No. 62-1) (“Steinman Third Rep.”); Report, dated January 22, 2022, filed as Ex. 71 (ECF No. 68-1) (“Steinman Fourth Rep.”). All told, Dr. Steinman prepared *more than 100 pages* in support of Petitioner’s claim (although many are either devoted to the disputed GBS diagnosis, or reflect reproduction of charts and wholesale sections of a variety of secondary sources he has relied upon).

As shown in his CV, Dr. Steinman received his undergraduate degree from Dartmouth College, and his medical degree from Harvard Medical School. *Curriculum Vitae*, filed as Ex. 16 on July 16, 2020 (ECF No. 38-2) (“Steinman CV”) at 1. He then completed residencies in neurology and pediatrics at Stanford University. *Id.* He has worked as a professor of neurology and pediatrics at Stanford for the past 40 years. *Id.*; Steinman First Rep. at 1. He is board certified in neurology from the American Board of Psychiatry and Neurology. Steinman CV at 2. Dr. Steinman has also published hundreds of peer-reviewed publications on neurology and autoimmune diseases. *Id.* at 6–49. He holds several patents related to the diagnosis and treatment of autoimmune and demyelinating diseases. *Id.* at 2–3. He presently serves as the George A. Zimmerman Professor of Neurological Sciences, Neurology, Genetics and Pediatrics at Stanford University. *Id.* at 1.

First Report

After several pages discussing his credentials, Dr. Steinman’s first report summarized the medical records, accepting Petitioner’s GBS diagnosis. Steinman First Rep. at 4–7. He also provided an overview of GBS, denying that Petitioner’s preexisting HIV was a likely explanation for his injury. *Id.* at 8–9.

With respect to causation, Dr. Steinman proposed that the pneumococcal vaccine contains a “molecular mimic” relevant to the pathogenesis of GBS, the “polar head group of the phosphoglycerol and phosphocholine molecules.” First Steinman Rep. at 11. Thus, his theory parallels what is often provided in many Program cases: that the mechanism of molecular mimicry—in which antibodies produced in reaction to a vaccine’s antigenic components cross-react with/mistakenly attack self structures in the body that resemble the antigens—was the foundational explanation for how the pneumococcal vaccine would also lead to GBS.

Of course, Dr. Steinman needed to show how molecular mimicry would specifically apply in this context, and he endeavored to do so. As the “Pneumovax-13” package insert indicates,⁴ the vaccine comprises a sterile suspension of saccharides of the capsular antigens of *Streptococcus pneumoniae* from 13 strains, or serotypes (1, 3, 4, 5, 6A, 6B, 7F, 9V, 14, 18C, 19A, 19F and 23F), individually linked to non-toxic diphtheria CRM₁₉₇ protein.⁵ *Id.* at 10–13 (citing Pneumococcal Package Insert, filed as Ex. 25 on July 16, 2020 (ECF No. 40-9) (“Pneumococcal Package Insert”), at 24);⁶ see generally D. Wang et al., *Uncovering Cryptic Glycan Markers in Multiple Sclerosis (MS) and Experimental Autoimmune Encephalomyelitis (EAE)*, *Drug Dev. Res.* 172, 185–86 (2014), filed as Ex. 31 (ECF No. 41-5). Certain phospholipids (phosphatidyl serine and phosphatidyl choline) are also present in the vaccine, expressed in association with its polysaccharide antigens. Steinman First Rep. at 12. In addition, the CRM₁₉₇ protein conjugate requires functional glycerol phosphate “side chains” be retained in the vaccine’s formulation (for the conjugate to perform its immune-boosting task). *Id.* at 13.

At the same time, Dr. Steinman stressed (invoking his own personal research in support) that “phospholipids are components” of the nerve myelin, and are moreover “targeted by antibodies in neuroinflammation.” First Steinman Rep. at 13–14; P. Ho et al., *Identification of Naturally Occurring Fatty Acids of the Myelin Sheath That Resolve Neuroinflammation*, 4 *Sci. Translational Med.* 1, 1 (2012), filed as Ex. 22 (ECF No. 27-1) (“Ho”). Ho, however, did not involve GBS specifically, but multiple sclerosis (“MS”)—a demyelinating autoimmune disease of the central nervous system—and although it suggests the studied antibodies might *contribute* to MS’s pathogenesis, it did not determine that they were the likely instigating spark for an autoimmune disease. *Compare* Ho at 1 with First Steinman Rep. at 17–18.

⁴ Pneumovax-13 is a trade name for a form of the pneumococcal vaccine that is covered under the Vaccine Act.

⁵ “CRM₁₉₇ is a nontoxic variant of diphtheria toxin isolated from cultures of *Corynebacterium diphtheriae* strain Cy (β197) grown in a casamino acids and yeast extract-based medium. CRM₁₉₇ is purified through ultrafiltration, ammonium sulfate precipitation, and ion-exchange chromatography.” Pneumococcal Package Insert at 24. It is included to induce “immunity through a T cell-dependent response.” *Deshler v. Sec’y of Health & Hum. Servs.*, No. 16-1070V, 2020 WL 4593162, at *19 (Fed. Cl. Spec. Mstr. July 1, 2020).

⁶ Also cited as Respondent’s Ex. M, Tab 5.

Dr. Steinman offered several other items of literature to support the conclusion that “phosphosaccharides with the same polar head groups are critical for the immunogenicity” of the vaccine. First Steinman Rep. at 15; J. Chang et al., *Relevance Of O-Acetyl and Phosphoglycerol Groups for the Antigenicity of Streptococcus Pneumoniae Serotype 18C Capsular Polysaccharide*, 30 Vaccine 7090, 7091 (2012), filed as Ex. 32 (ECF No. 41-6) (“Chang”) (demonstrating that a phospholipid linkage is necessary for immunogenicity of these capsular polysaccharides); C. Lugowski & H. Jennings, *Structural Determination of the Capsular Polysaccharide of Streptococcus Pneumoniae Type 18C*, 131 Carbohydrate Res. (1984), filed as Ex. 44 (ECF No. 68-4) (same); J. Ohori et al., *Phosphorylcholine Intranasal Immunization with a 13-Valent Pneumococcal Conjugate Vaccine can Boost Immune Response Against Streptococcus Pneumoniae*, 38 Vaccine 699, 699 (2020), filed as Ex. 24 (ECF No. 41-8) (“Ohori”); Yi-Ping Chuang et al., *Impact of the glpQ2 Gene on Virulence in a Streptococcus pneumoniae Serotype 19A Sequence Type 320 Strain*, 83 Infection & Immunity 682, 682 (2015), filed as Ex. 28 (ECF No. 27-7) (“Chuang”).

Chang, for example, was offered to establish the immunologic significance of the serotype 18C polysaccharide found in the vaccine’s antigenic components, in order to demonstrate that “[t]he phosphoglycerol linkage is critically preserved to ensure the vaccine’s function.” First Steinman Rep. at 15–16; Chang at 7095. This is, however, not a precise construction of Chang’s findings. It is true that the pneumococcal vaccine must, to be effective, *induce* the production of anti-capsular polysaccharide antibodies. Chang at 7090. However, the *S. pneumoniae* bacteria’s polysaccharide antigens can have their immunogenicity affected by the manner in which the conjugated version of the vaccine (deemed the “vaccine of choice to target child protection”) is manufactured/formulated. *Id.* Chang’s authors therefore conducted an animal study in which the relevant capsular polysaccharide used in most versions of the vaccine was treated and prepared in different ways, then injected into the animal subjects, in order to evaluate “a suitable modification-conjugation procedure” that would ensure preservation of the most important antigenic features of the vaccine. *Id.* at 7095; *see also* 7091–92. Chang concluded that preservation of the glycerol phosphate group was more important than the O-acetyl group. *Id.* at 7095–96. Thus, Chang says *nothing about those antigens being pathogenic*, but only that they must *exist* in the vaccine if it is to perform as intended.

Dr. Steinman also maintained that Ohori showed “[a]dditional [intranasal] immunization with [phosphorylcholine (“PC”)] after PCV13 immunization, which is currently conducted under a periodic vaccination program, can produce a booster effect comparable to that achieved by additional systemic immunization as well as PC-specific mucosal immune response, thereby providing protection against *S. pneumoniae* serotypes not contained in PCV13.” First Steinman Rep. at 16–17; Ohori at 699. Ohori’s focus, however, was on establishing ways to improve the body’s immune response to the intramuscularly-administered pneumococcal vaccine—here, by supplementing it with phosphorylcholine administered intranasally, after initial vaccination. Ohori

at 700 (“the transmucosal administration of PC can induce systemic as well as mucosal immune responses and thus provide a higher preventative efficacy compared with conventional vaccines”). Thus, while Ohori may confirm that the vaccine *contains* PC, it says nothing at all about how the presence of it could prove pathogenic.

Dr. Steinman did not, however, merely speculate that the putatively-pathogenic antibodies should be found in the vaccine—he concluded that in fact they “ARE.” First Steinman Rep. at 19 (capitalization in original), *citing* Chuang. In particular, Dr. Steinman maintained, Chuang demonstrated that the phospholipid phosphorylcholine is expressed in the 19A serotype component, which is key in the pathophysiology of the pneumonia infection. Steinman First Rep. at 19. The enzyme for metabolizing lipids and producing phosphorylcholine was also present in strains 3, 6B, 19A, and 19F—with three of those strains (strains 3, 19A, and 19f) all included in the Prevnar formulation. Chuang at 682.

Another article was referenced by Dr. Steinman to show more specifically that the antibodies of the kind that would (theoretically) be produced by the pneumococcal vaccine are known to be associated with GBS. B. Gilburd et al., *Autoantibodies to Phospholipids and Brain Extract in Patients with Guillain-Barre Syndrome: Cross-Reactive or Pathogenic?* 16 *Autoimmunity* 1, 23–27 (1993), filed as Ex. 27 (ECF No. 41-1) (“Gilburd”); First Steinman Rep. at 18. However (and ignoring that Gilburd was published nearly 30 years ago, yet does not appear to have encouraged much, if anything, in the way of follow-up research), the article is more notable for its caveats and limitations. Thus, Gilburd admits at its outset that “the autoantigen of GBS has not yet been identified”—still a truism so many years later. Gilburd at 23. Further, its authors note something even more fundamental—that “it is not settled whether the autoantibodies in GBS induce the nerve damage *or are induced by the demyelination and liberation of autoantigens.*” *Id.* (emphasis added). Otherwise, Gilburd’s findings were based on testing involving an exceedingly small sample (16 GBS patients), and its authors reiterated the uncertainty as to what the temporal role was for these autoantibodies. *Id.* at 24, 26–27.

The same qualifications apply to a case report filed by Dr. Steinman. First Steinman Rep. at 18–19; G. Nakos et al., *Anti-Phospholipid Antibodies in Serum from Patients with Guillain-Barré Syndrome*, *Intensive Care Med.* 1401, 1404 (2005), filed as Ex. 28 (ECF No. 41-2) (“Nakos”). Nakos, Dr. Steinman maintained, observed that “phospholipid antibodies were found in patients with GBS.” First Steinman Rep. at 18. In Nakos, a small sample of nine patients with the AIDP (meaning “acute inflammatory demyelinating polyneuropathy”) GBS variant had their blood tested for the presence of anti-phospholipid antibodies over a period of several days, with results revealing “a wide range” of these antibodies in comparison to controls. Nakos at 1405. Nakos did observe phosphatidylcholine (which has a phosphoglycerol component) as one of the main antigens in the tested sample. *Id.* at 1405–07. But Nakos’s authors did not purport to determine the precise *role* of the autoantibodies in GBS’s pathogenesis, allowing that they could

simply “represent a part of a more extensive immunoreaction that takes place in the GBS,” rather than a primary/initial causal factor. *Id.* at 1406. Thus, evidence of the *presence* of the putatively-pathogenic antibody was not the same as evidence it *is* pathogenic.

Dr. Steinman's own research, he added, had shown that phospholipids are components of the nerve's myelin sheath in humans, and that they are targeted by antibodies in the context of existing neuroinflammation. Steinman First Rep. at 7; J. Kanter et al., *Lipid Microarrays Identify Key Mediators of Autoimmune Brain Inflammation*, 12 Nat. Med. 138 (2005), filed as Ex. 21 (ECF No. 26-8) (“Kanter”). Employing an experimental animal model, Kanter⁷ determined that (given the extent to which nerve myelin is comprised of lipids) lipid-specific autoantigens likely played *some* role in the pathogenesis of MS—although Kanter is not specific to peripheral neuropathies like GBS. Kanter at 138, 142. It also did not suggest this role was initial or primary. To the contrary, Kanter's authors also noted that prior studies had suggested that the lipid-oriented attacks might be *secondary* to ongoing autoimmune-mediated damage, occurring as a result of “epitope spreading” encouraged by other antibodies. *Id.* at 141.

Finally, Dr. Steinman endorsed the onset of Mr. Gamboa's GBS as having occurred in a medically-acceptable timeframe when measured from date of vaccination. First Steinman Rep. at 20. For support, he invoked an item of literature specific to the 1976 swine flu vaccine (and something he has cited in virtually every single one of his expert reports involving neuropathies that I have reviewed since becoming a special master). L. Schonberger et al., *Guillain-Barré Syndrome Following Vaccination in the National Influenza Immunization Program, United States, 1976–1977*, 110 Am. J. Epidemiol. 105 (1979), filed as Ex. 30 (ECF No. 27-9) (“Schonberger”). Because Petitioner's onset occurred approximately nine days after vaccination, it fell within Schonberger's observed risk interval. Schonberger at 109. In so maintaining, Dr. Steinman proposed that what is known about timing in Schonberger could be applied herein, although he also admitted that more detailed studies specific to GBS and the pneumococcal vaccine do not exist. Steinman First Rep. at 9.

Second Report

Dr. Steinman's next report reacted to the reports filed by each of Respondent's experts—although for present purposes only his efforts to rebut Dr. Whitton's opinion merit discussion. *See generally* Second Steinman Rep. at 1–14. First, he contended that in fact *S. pneumoniae* is associated with GBS. *Id.* at 1–3. But he offered only two case reports to substantiate this contention. H. El Khatib et al., *Case Report: Guillain-Barré Syndrome With Pneumococcus – A New Association in Pediatrics*, 11 IDCases 36 (2018), filed as Ex. 40 (ECF No. 56-1) (“El Khatib”) (13 year old GBS patient was discovered to be suffering from existing case of acute pneumonia);

⁷ Kanter is another MS-specific study involving several of Ho's co-authors (in fact, Kanter pre-dates Ho and was cited therein). *See* Ho at 2 n.3.

N. Yuki & K. Hirata, *Fisher's Syndrome and Group A Streptococcal Infection*, 160 J. Neurol. Sci. 64 (1998), filed as Ex. 41 (ECF Mo. 56-2) (17 year-old presenting with what was later determined to be streptococcal tonsillitis later developed Miller-Fisher GBS variant).

Neither case report corroborates a purported association, however. It is, for example, not self-evident that the El Khatib subject even had GBS, since the patient had an intercurrent wild infection. Thus, El Khatib demonstrates that the individual in question had *Streptococcus* bacteremia from an ongoing bacterial infection, rather than a post-infectious condition that could be reasonably analogized to a vaccination. El Khatib at 37. And both case report's authors admitted that no such association had even been scientifically established. *See, e.g.*, El Khatib at 36 ("*Streptococcus pneumoniae* . . . has never been associated with GBS in the pediatric age group"). Even Dr. Steinman acknowledged that the association was less common than *C. jejuni*. Second Steinman Rep. at 3.

Second, Dr. Steinman endeavored to rehabilitate his prior argument that the pathologic process leading to GBS involves the targeting of phospholipid structures in the myelin. *See generally* Second Steinman Rep. at 3–9. In reaction to Dr. Whitton's point that literature (like Nakos) suggested that the anti-phospholipid antibodies might not be directly causal of GBS, but instead be generated by the disease process itself, Dr. Steinman protested that this was "not inconsistent" with his theory. *Id.* at 3. His point, rather, was that (a) these antibodies "are found" in GBS, and (b) that they could theoretically be generated by the vaccine. *Id.* However, Dr. Steinman nevertheless insisted they *did* likely play a role in direct disease pathogenesis. *Id.* (citing Ho ("antibodies from patients with multiple sclerosis target the phosphate polar head group (the phosphoglycerol and phosphocholine moiety) in the phospholipids of the central nervous system"). And he maintained that it was reasonable to rely on items of literature, like Ho or Kanter, that focused on MS rather than peripheral neuropathies like GBS, given the "similarity of the antibody targets" in both diseases. Second Steinman Rep. at 5.

Relatedly, Dr. Steinman attempted to establish that the polar head groups could also, like gangliosides (about which far more is known, it must be underscored), be the specific target for the autoimmune cross-reactive processes leading to GBS. Second Steinman Rep. at 5–8. He contested Dr. Whitton's argument about the specificity of different kinds of bacterial structures (and that some were less likely to express ganglioside mimics), maintaining that pneumococcus was not ruled out despite its structure (although he offered only his case reports, like El Khatib, as evidence of this). *Id.* at 5. He also again stated that studies had shown the presence of the antibodies to phospholipids in the blood sera of GBS patients. *Id.* at 6–7 (citing Nakos).

More specific to the point at issue, Dr. Steinman maintained that Dr. Whitton's analysis of the nature of an antibody attack on the polar head groups misconstrued his actual argument—which was that the polar heads *themselves* were a target, as opposed to the "side chains that adorn

the phosphoglycerol or the phospholipid molecule as a whole.” Second Steinman Rep. at 6. In support, he referenced Ho. *See* Ho at 3–4 (“[t]his suggests that autoantibodies present in RRMS CSF target the phospholipids’ phosphate head group and that the affinity of antibody-lipid binding is not specific to a particular phospholipid.”). Thus, even though Dr. Steinman conceded that “gangliosides generally do not contain polar head groups,” and gangliosides might be “important” in GBS, they were not necessarily the sole disease attack point/trigger (although his substantiation for this point was quite thin). Second Steinman Rep. at 7–9.⁸

Third Report

For his next supplemental report, Dr. Steinman opted to write more than 40 pages—almost the total length of the first two reports put together (and raising the obvious question of why a responsive report would *ever* need to be so long).⁹ Although Dr. Steinman again used the opportunity to react to both of Respondent’s experts and their criticisms, three-fourths of the report was aimed at addressing Dr. Whitton.

Dr. Steinman first sought to explain why studies involving MS, which he had relied on in his earlier reports (*see, e.g.*, Kanter, Ho) could have applicability in the context of GBS. Third Steinman Rep. at 2–3. He emphasized his expertise researching “[h]ow the immune system attacks the nervous system,” but otherwise repeated his conclusion that what was seen in the MS context likely applied to GBS as well. *Id.* at 2. At most, he admitted that MS’s chronic nature made it somewhat easier to study.

⁸ Dr. Steinman also engaged in a lengthy effort to bulwark his prior argument that one of the vaccine’s strains contains glycerol phosphate, against Dr. Whitton’s contention that it does not. Second Steinman Rep. at 10–11. In so doing, he noted that articles like Chang established that other strains in the vaccine could also be shown to contain a phosphoglycerol group, and that its inclusion was important, because it ensured the vaccine’s function. *Id.* at 11–12; Chang at 7095. Dr. Whitton also engaged in this debate in his subsequent reports, protesting that Dr. Steinman was flat-out wrong. *See, e.g.*, Second Whitton Rep. at 4–5.

I do not highlight this dispute (or attempt to parse which expert ended up “on top” with respect to the particulars of this sub-debate), because my *Althen* prong one determination turns more on questions about the general associations between the vaccine and GBS, as well as the known pathogenic course of GBS—factors that are preponderantly unresponsive of Dr. Steinman’s theory, *even if* he is correct that the pneumococcal vaccine’s effectiveness depends in part of phospholipid molecular structures, or that enough serotypes of the *S. pneumoniae* bacteria included in the vaccine contain phosphoglycerol molecules.

⁹ Of course, much of this wholly-excessive length was not due to the inclusion of actual expert thought or comment. Rather, and as is common with Dr. Steinman’s expert reports in Program cases, this third report was chock-full of reproductions of charts or intricate molecular structure images taken from his source materials. Indeed, by my admittedly-cursory count, the third report alone contains more than 15 chart or figure reproductions (not to mention numerous lengthy block-quotes from his own prior reports or Dr. Whitton’s). *See generally* Third Steinman Rep. at 1–30.

Second, Dr. Steinman again ventured to show how the pneumococcal bacterium could be associated with GBS, despite Dr. Whitton's contention that the most reliable authority never included it in lists of likely antecedent triggers. Third Steinman Rep. at 3–6. He specifically referenced one such article. *See generally* A. Jasti et al., *Guillain-Barre Syndrome: Causes, Immunopathogenic Mechanisms and Treatment*, 12 Expert Rev. Clinical Immunology 1175 (2016), filed as Ex. A, Tab 17 (ECF No. 45-7) (“Jasti”). Although, Dr. Steinman admitted, Jasti provided “a long list” of potential triggers for GBS that did not also include *S. pneumoniae*, it also supported the conclusions that (a) molecular mimicry was the most likely mechanistic explanation for how *any* trigger would lead to GBS, and (b) that a wide array of external insults (viral as well as bacterial) could cause GBS. (He also made some less-well-supported contentions about Jasti, such as representing that it demonstrated gangliosides were not the only possible target for cross-reaction. Jasti at 1184).¹⁰

From this, Dr. Steinman moved to a slightly different contention: that the pneumococcal vaccine could theoretically cause GBS *even if* a natural pneumococcal infection could not, reasoning that “the vaccine is very different from the microbe itself.” Third Steinman Rep. at 3. In so arguing, he noted that the vaccine did not merely contain bacterial serotypes, but was conjugated to the non-toxic diphtheria CRM₁₉₇ protein, to improve its immunogenicity for its targeted “audience” (young children and the elderly). *Id.* at 4–5. But that protein was only slightly different from the diphtheria toxin. *Id.* at 4.

Later in his third report, Dr. Steinman explained this contention in more detail. Third Steinman Rep. at 17–18, 23–29. All 13 of the vaccine's polysaccharide serotypes are “covalently linked” to the CRM₁₉₇ conjugate (which Dr. Steinman described as “reductive amination”). *Id.* at 18. This was necessary to preserve “the chemical structure of the phosphoglycerol moiety.” *Id.* But in addition to providing this function, the conjugate *also* contained an *additional* mimic that could cause GBS (although he deemed it not mutually exclusive to the primary antigenic, phosphoglycerol-containing components that his prior reports had focused on). *Id.* at 24.

Specifically, the conjugate includes Contactin-1, a molecule “that is targeted in some cases of GBS.”¹¹ Third Steinman Rep. at 24; Y. Miura et al., *Contactin I IgG4 Associates to Chronic Inflammatory Demyelinating Polyneuropathy with Sensory Ataxia*, 138 Brain 1484, 1486 (2015), filed as Ex. 56 (ECF No. 63-10) (“Miura”) (aiming to describe the clinical and serological features

¹⁰ For good measure, Dr. Steinman threw in the proposition that the Petitioner had tested *negative* for anti-ganglioside antibodies. Third Steinman Rep. at 3. To some extent, however, this raises a question relevant to the second *Althen* prong, and hence I do not discuss it. If the pneumococcal vaccine *cannot likely* cause GBS, it does not matter what kinds of antibodies Petitioner was shown by testing to possess (or not).

¹¹ Contactin-1 is a molecule that some studies have identified as a target in the myelin damage central to GBS. *Trollinger v. Sec'y of Health & Hum. Servs.*, No. 16-473V, 2023 WL 2521912, at *8 (Fed. Cl. Spec. Mstr. Feb. 17, 2023), *mot. for review den'd*, No. 16-VV-473, 2023 WL 5249583 (Fed. Cl. July 31, 2023).

of Japanese patients with CIDP displaying the anti Contactin-1 antibodies). A different article, Dr. Steinman proposed, established the possibility that the conjugate *could* express a mimic for Contactin-1. Third Steinman Rep. at 29; R. Raju et al., *Epitopes for Human CD4+ Cells on Diphtheria Toxin: Structural Features of Sequence Segments Forming Epitopes Recognized by Most Subjects*, 25 Eur. J. Immunology 3207, 3207 (1995), filed as Ex. 55 (ECF No. 63-9) (“Raju”) (focusing on the generation of T helper cells in reaction to diphtheria toxin—not showing that antibodies would cross react with Contactin-1).

To identify the possible molecular congruence, Dr. Steinman conducted a “BLAST” search¹² focusing on Contactin-1 in comparison with the conjugate’s components. Third Steinman Rep. at 24. He thus performed the same desktop computer analysis he has offered in countless Program cases,¹³ where he seeks to establish the potentiality of molecular mimicry. Specifically, he (a) attempted to propose the degree of amino acid homology that would be necessary, arguing that five out of a twelve amino acid string is sufficient (*Id.* at 25) (b) performed searches with the BLAST online government database for the components of the pneumococcal vaccine, to identify the amino acids comprising them, and determine whether other researchers identified the same epitope(s)¹⁴, and (c) compared them to the molecular composition in identified portions of myelin. *Id.* at 25–29. He concluded from the foregoing that “a compelling theory” existed in support of his contention that even based on the conjugate itself, “molecular mimics in the [pneumococcal vaccine] can cause GBS.” *Id.* at 29.

Dr. Steinman also sought to add heft to his prior contention that the vaccine could likely induce cross-reactive antibodies specific to neuronal polar head groups found in or on the nerve myelin. Third Steinman Rep. at 8–13. He stressed that the vaccine contained phosphoglycerols in at least some of its included polysaccharide strains, and that they played an important role in ensuring immunogenicity. *Id.* at 8–9, 14–15, *citing* Chang at 7095. Because (as established in Chang) the phosphoglycerol structure was important to the bacteria’s ability to cause infection in

¹² Basic Local Alignment Search Tool (“BLAST”) is a medical/scientific internet resource that assists researchers in finding regions of similarity between biological sequences of amino acids. The program compares nucleotide or protein sequences to sequence databases and calculates the statistical significance. BLAST, U.S. National Library of Medicine, <https://blast.ncbi.nlm.nih.gov/Blast.cgi> (last visited Feb. 17, 2023). A BLAST search involves review of an online database to “compare[] nucleotide and protein sequences, to search for a homology between the . . . vaccine and [the body’s myelin basic protein].” *Montgomery v. Sec’y of Health & Hum. Servs.*, No. 15-1037V, 2019 WL 2511352, at *5 (Fed. Cl. Spec. Mstr. May 21, 2019).

¹³ *Schilling v. Sec’y of Health & Hum. Servs.*, No. 16-527V, 2022 WL 1101597, at *5 (Fed. Cl. Spec. Mstr. Mar. 17, 2022) (“[Dr. Steinman] also (as he has done many times before) performed online BLAST searches to identify amino acid sequence homology. . .”).

¹⁴ Dr. Steinman first tested the sequence WEQAKALSVE and identified it as an area of alignment between contactin-1 and CRM₁₉₇, determining it is “an epitope in diphtheria toxin, which provide the basis for CRM₁₉₇.” Steinman Third Rep. at 28. Dr. Steinman tested a second sequence, EYMAQACAGNRVRR, which also has “known cross-reactivity with epitopes described in humans and on the c. diphtheria microbe that is the basis for CRM₁₉₇.” *Id.* at 29.

the lung, it was also a “critical target” for an immune response intended (due to the vaccine) to arrest the bacterial progress. Third Steinman Rep. at 16; Chang at 7090, 7095.

Other literature more specifically demonstrated, in Dr. Steinman’s estimation, the degree to which the pneumococcal vaccine’s phosphoglycerol components likely sparked a directed immune response. Third Steinman Rep. at 17; Bryson et al., *Structures of Preferred Human IgV Genes-Based Protective Antibodies Identify How Conserved Residues Contact Diverse Antigens and Assign Source of Specificity to CDR3 Loop Variation*, 196 J. Immunology 4723, 4725, 4727–28 (2016), filed as Ex. 65 (ECF No. 79-4) (“Bryson”). Bryson, he explained, showed that the immune response to the vaccine “targets” phosphoglycerol in serotype 23F. Third Steinman Rep. at 9–10, 12 (deeming the evidence from Bryson on this point to “UNEQUIVOCALLY” support his contention (emphasis in original)); Bryson at 4723–24. Further, the fact that phosphoglycerol was not present for all 13 of the vaccine’s serotypes did not make a difference to Dr. Steinman. The vaccine, he maintained, was intended to generate an individual response to *each* separate serotype, and thus only one serotype including phosphoglycerol was needed to generate the antibodies against the polar heads. Third Steinman Rep. at 13.

Otherwise, Dr. Steinman reiterated points he had already covered in his two prior efforts. For example, he maintained that epidemiologic studies might establish the vaccine’s general safety, but could not disprove the possibility of vaccine-caused injury. Third Steinman Rep. at 6–7; P. Haber et al., *Post-Licensure Surveillance of 13-Valent Pneumococcal Conjugate Vaccine (PCV13) in Adults Aged 19 Years Old in the United States, Vaccine Adverse Event Reporting System (VAERS), June 1, 2012–December 31, 2015*, 34 Vaccine 6330, 6330, 6333–34 (2016), filed as Ex. M, Tab 7 (ECF No. 78-7) (“Haber”); H. Tseng et al., *Pneumococcal Conjugate Vaccine Safety in Elderly Adults*, Open Forum Infectious Diseases 1, 1 (2018), filed as Ex. M, Tab 8 (ECF No. 78-8) (“Tseng”). Tseng, in fact (in Dr. Steinman’s reading) revealed what he deemed an unexplained “high incidence” of GBS for those receiving the pneumococcal vaccine versus an expected background rate (four cases in a 1–42 period for a sample of 313, 136 vaccinated individuals, versus one case for 78,284 individuals). Tseng at 7.

Fourth Report

Dr. Steinman’s final written report focused more on Dr. Whitton’s rejoinders than commenting on Dr. Chaudhry’s final opinions. *See generally* Fourth Steinman Rep. at 1–14.

Dr. Steinman relied on some recent studies, one of which he co-authored, to bulwark the mechanistic aspect of his theory. *See* Fourth Steinman Rep. at 1, 5–6; W. Robinson & L. Steinman, *Epstein-Barr Virus and Multiple Sclerosis*, 21 Science 375:264 (2022), filed as Ex. 72 (ECF No. 68-2) (“Robinson & Steinman”); T. Lanz et al., *Clonally Expanded B Cells in Multiple Sclerosis Bind EBV EBNA1 and GlialCAM*, 603 Nature 1 (2022), filed as Ex. 73 (ECF No. 68-3) (“Lanz”).

Of course—and as Dr. Steinman conceded—these articles relate *solely* to MS and a possible disease-driving mimic between self amino-acid sequences and the Epstein-Barr virus (“EBV”), and thus specifically *say nothing about a different vaccine and different disease*. Fourth Steinman Rep. at 1; Lanz at 1; Robinson & Steinman at 1. However, Dr. Steinman deemed them relevant because they undermined the degree of homology Dr. Whitton had argued was necessary for a likely cross-reaction. Fourth Steinman Rep. at 5–6.

Otherwise, Dr. Steinman’s fourth report simply repeated his prior contentions in the tit-for-tat debate between him and Dr. Whitton evident from their prior written exchanges. He again argued that MS-specific studies had evidentiary value, summarizing the findings from articles previously discussed, like Ho (which showed polar head targeting in MS), and connecting them to Nakos (which he now maintained revealed “more antibody to phospholipid than to ganglioside in patients with GBS”). Fourth Steinman Rep. at 1–2. He repeated the same comparison, while adding in references to Chang, Chuang, and Bryson, to defend the contention that the phosphoglycerol-specific antibodies could cross-react with polar group heads, stressing again the point that reliable science showed the existence of phosphoglycerol in the 23F serotype strain. *Id.* at 2–4.¹⁵ Lastly, Dr. Steinman took on directly Dr. Whitton’s criticisms of his data search methodology and analysis (discussed below), arguing that it had scientific reliability and merit. *Id.* at 4–14.

B. Respondent’s Expert¹⁶ – *J. Lindsay Whitton, M.D., Ph.D.* – Dr. Whitton, an immunologist, submitted three expert reports for the Respondent in support of the argument that the pneumococcal vaccine does not likely cause GBS. Report, dated September 2, 2020, filed as Ex. A (ECF No. 44-1) (“First Whitton Rep.”); Report, dated August 3, 2021, filed as Ex. G (ECF No. 61-1) (“Second Whitton Rep.”); Report, dated November 23, 2021, filed on November 23, 2021 (ECF No. 67-1) (“Third Whitton Rep.”). Dr. Whitton disputed the contention that the phosphoglycerol/glycerophosphate-containing polysaccharides in certain pneumococcal vaccine antigens could induce an antibody response that causes GBS, or that the conjugate could similarly spark an autoimmune attack.

Dr. Whitton obtained his undergraduate, medical, and doctorate degrees from the University of Glasgow in Scotland. *Curriculum Vitae*, filed as Ex. B on September 2, 2020 (ECF No. 45-12) (“Whitton CV”) at 1; Whitton Rep. at 1. He then began working as a senior research associate at the Scripps Research Institute in La Jolla, California, where he studied immunology, vaccinology, and viral pathogenesis. Whitton CV at 1; Whitton Rep. at 1. Dr. Whitton has conducted extensive research in these subject areas and has published numerous articles on the

¹⁵ He also copied wholesale charts and images in the final report from his earlier offerings. *Compare* Third Steinman Rep. at 10–11 *with* Fourth Steinman Rep. at 3–4.

¹⁶ Although diagnostic expert, Dr. Vinauy Chaudhry, also prepared several written reports, I do not summarize them because of my focus herein on the first causation prong.

subjects. Whitton CV at 2–15. In addition to his research, Dr. Whitton also serves as a professor in the department of Immunology and Microbial Science at Scripps Research Institute. Whitton CV at 1. In both his research and teaching, Dr. Whitton has focused on viral pathogenesis, innate and adaptive immune responses, and molecular mimicry. Whitton Rep. at 1–2. Dr. Whitton does not have board certification in the United States to practice medicine. *Id.* at 2.

Dr. Whitton began with a lengthy discussion of the pneumococcal vaccine itself. First Whitton Rep. at 3–6. The vaccine aims to immunize against a bacterial infection caused by *S. pneumoniae*, or “pneumococcus.” *Id.* at 3. The *S. pneumoniae* bacterium is surrounded by a “capsule” made of polysaccharides. *Id.* at 3–4. Polysaccharides are made up of sugar molecules and occur throughout nature, but are distinguishable from a protein (which is comprised of chains of amino acids). *Id.* Amino acids contain only two attachment points, so amino acid chains have a linear character. *Id.* at 4. Sugars, on the other hand, have several different points at which they can link, resulting in different molecular “shapes.” *Id.*; J. Prestegard et al., *Oligosaccharides and Polysaccharides*, Essentials of Glycobiology 1, 4 (2017), filed as Ex. A, Tab 1 (ECF No. 44-2). Thus, because of such variation, the pneumococcal vaccine can only “teach” the immune system to respond to the specific strains it “sees” (based on the strains in the vaccine), with each being separate. First Whitton Rep. at 5. There are close to 100 serotypes¹⁷ of pneumococcus, and they are distinguished based on having structurally-different polysaccharide capsules. *Id.*

In addition, Dr. Whitton explained the “conjugated” character of the version of the pneumococcal vaccine at issue—Pneumovax-13. Although adults can mount a robust immune response when exposed simply to the bacterial polysaccharides alone, it has been found that children (especially infants) do not. First Whitton Rep. at 5. As a result, to boost the immune response (and in particular the intended production of antibodies to the *S. pneumoniae* bacterium), the vaccine’s antigenic components are linked, or conjugated, to a protein that “can trigger a reasonably strong T cell response” (and in this case Dr. Whitton referred to “helper T cells”—lymphocytes that assist B cells in production of antibodies specific to the vaccine’s polysaccharide antigens).¹⁸ *Id.* at 6.

Only the conjugated version of the pneumococcal vaccine is “covered” by the Vaccine Program,¹⁹ and many studies have noted not only its general safety, but the fact that it (or at least

¹⁷ Dr. Whitton defined serotype to mean “this organism is distinguished from related organisms on the basis of how it is recognized by the antibody response.” First Whitton Rep. at 5 (emphasis in original).

¹⁸ For a more detailed discussion of the CRM₁₉₇ conjugate’s function, see *Bielak v. Sec’y of Health & Hum. Servs.*, No. 18-761V, 2022 WL 18058244, at *31 (Fed. Cl. Spec. Mstr. Dec. 9, 2022).

¹⁹ Another version—Pneumovax 23—is not similarly conjugated, and because it is only recommended for adults, it cannot be the basis of a Program claim. Whitton Rep. at 6; *Louvaris v. Sec’y of Health & Hum. Servs.*, No. 21-416V, 2021 WL 4955690, at *1–2 (Fed. Cl. Spec. Mstr. Sept. 27, 2021)

the antigenic components common to both versions) is not associated with an increased risk of disease—including GBS. First Whitton Rep. at 10 (*citing* Haber). Dr. Whitton admitted that Haber relied on passive VAERS²⁰ reports (which are not verified for accuracy) rather than epidemiologically-verified data, but maintained that its findings still had value since the “early warning system” benefits provided by VAERS undercut the concept that GBS might have some relationship to the pneumococcal vaccine. He also noted that Dr. Steinman’s report had itself cited a large, proper epidemiologic study that reached the same conclusions. First Whitton Rep. at 10; R. Baxter et al., *Lack of Association of Guillain-Barre Syndrome with Vaccinations*, 57 *Clinical Infection Diseases* 197, 201 (2013), filed as Ex. 37 (ECF No. 42-1) (“Baxter”). Although Baxter had only considered the non-covered version of the pneumococcal vaccine, Dr. Whitton still deemed its findings significant, since that vaccine (like Prevnar-13) did not contain the antigenic molecules (discussed below) that he deemed more likely to be capable of causing GBS.

Dr. Whitton also included in his first report some discussion of GBS—and in particular the pathogenic mechanisms most likely to trigger it. *See generally* First Whitton Rep. at 7–10. GBS was known to be associated more often than not with a pre-onset infection of some kind, and was believed to be mediated by some autoimmune process. *Id.* at 7; J. Winer et al., *A Prospective Study of Acute Idiopathic Neuropathy – Antecedent Events*, 51 *J. Neurology, Neurosurgery, & Psychiatry* 613 (1988), filed as Ex. A, Tab 12 (ECF No. 45-2). Antibodies, he agreed, were a more likely driver of the autoimmune cross-attack in GBS, with “host gangliosides” present in peripheral nerves their most well-understood target. P. van Doorn et al., *Clinical Features, Pathogenesis, and Treatment of Guillain-Barré Syndrome*, 7 *Lancet Neurol.* 939 (2008), filed as Ex. A, Tab 6 (ECF No. 44-7). Thus, anti-ganglioside antibodies observed in the blood sera of GBS patients likely played a “pathological role” in driving GBS. First Whitton Rep. at 7.²¹

²⁰ VAERS is a database maintained by the CDC to compile information from reports about reactions to immunizations listed on the Vaccine Injury Table, 42 U.S.C. § 300aa-14(a). *See About VAERS*, VAERS, <https://vaers.hhs.gov/about.html> (last visited Feb. 17, 2023).

²¹ In fact, Dr. Whitton maintained that Dr. Steinman had (in literature *he* co-authored) acknowledged that gangliosides were the most likely GBS autoimmune target in *any* case of GBS, as opposed to phosphoglycerol structures. First Whitton Rep. at 13, *citing* Kanter at 138 (“[a]utoimmune responses directed against phospholipids and gangliosides contribute to the pathogenesis in systemic lupus erythematosus and [GBS], respectively”). Dr. Whitton interpreted this sentence construction (and the word “respectively” at its end) to mean that the phospholipid target reference is *only* specific to lupus, not GBS—a wholly reasonable construction in my view. Dr. Steinman later denied this reading, however, protesting variously that it did not reflect the views of “all” of Kanter’s authors, or was otherwise a misreading. *See, e.g.*, Second Steinman Rep. at 8–9.

This is not the first time this expert pair has disputed the interpretation of this sentence from Kanter in the context of a claim that the pneumococcal vaccine can cause GBS. *Trollinger*, 2023 WL 2521912, at *4, 16. While (as before) I deem Dr. Whitton’s reading of the sentence to be more compelling and grammatically accurate, I do not also view this to be a full-on concession by Dr. Steinman about weaknesses in his theory. Rather, it simply underscores that science has *more consistently understood* gangliosides as the GBS target for *certain* autoimmune processes or triggers—and thus underscores the degree to which the alternative target proposed in this case is not all that well-supported by existing literature, even if it has some reasonable *theoretic* underpinnings. There would simply be *other* items of literature discussing GBS mediated by attacks on phosphoglycerol/phospholipid targets if the contention had reliable support.

GBS was also, Dr. Whitton acknowledged, understood to be associated with *some* kinds of infections. In particular, medical science was aware of the connection between *Campylobacter jejuni* bacteria (which typically cause some form of gastrointestinal disease) and GBS. First Whitton Rep. at 7–8. But Dr. Whitton stressed that the strains/serotypes in question still mattered—because not all bacteria (not even all strains of *C. jejuni*) expressed “lipo-oligosaccharides” (“LOS”), or “lipopolysaccharides” (“LPS”)—antigens capable of triggering the development of anti-ganglioside antibodies, due to their ganglioside-like structures. *Id.* at 8; I. Nachamkin et al., *Campylobacter Species and Guillain-Barré Syndrome*, 11 Clinical Microbiology Rev. 555, filed as Ex. A, Tab 18 (ECF No. 45-8). Thus, Dr. Whitton emphasized that “triggering of GBS by *C. jejuni* appears to be quite strain-specific, and appears to be determined by some aspect of the particular strain’s LPS molecule.” First Whitton Rep. at 8 (emphasis in original).

S. pneumoniae, by contrast, was not similarly associated with GBS. First Whitton Rep. at 8. Indeed, it was not included in larger lists of viral and bacterial infections so associated. *Id.*; Jasti at 1177–79 (discussing the causes and immunopathogenic mechanisms of GBS). This raised for Dr. Whitton a foundational weakness in Dr. Steinman’s theory: if the antigenic components of the vaccine could be causal of GBS, then why was there no recognized association with the underlying antigens from the actual infectious bacterial strains *included* in the vaccine? First Whitton Rep. at 9.

This in turn, Dr. Whitton opined, illuminated a second limitation for a causal theory proposing that antigens derived from *S. pneumoniae* bacterial strains could be just as causal of GBS as distinguishable bacteria like *C. jejuni*. Some bacteria known to be associated with GBS, like *C. jejuni*, are “gram negative,”²² (meaning their poly saccharide capsule is “thin”) but the *S. pneumoniae* capsule is “gram positive,” or thick. First Whitton Rep. at 9. Gram-negative bacteria include an outer membrane in which is “embedded” the LOS or LPS molecules believed to be capable of triggering autoantibodies against ganglioside structures—the pathogenic process leading to GBS. *Id.* Indeed, all but one of the wild bacteria already understood to be associated with GBS are gram negative. *Id.* at 9–10; Jasti at 1176. Thus, the *S. pneumoniae* bacterium (and the vaccine containing its antigenic strains) “lack the very molecules that are thought to trigger GBS.” First Whitton Rep. at 9 (emphasis in original).

Dr. Whitton then turned to the primary contentions contained in Dr. Steinman’s first report. First, he repeated his point that Dr. Steinman had made no effort to explain how causality was

²² Bacteria can be subdivided into two groups based on the nature of their cell walls, using a procedure called gram staining to distinguish the groups. First Whitton Rep. at 9. Gram-positive bacteria have thick capsules with no outer membrane. *Id.*; G. Zhang et al., *On the Essentiality of Lipopolysaccharide to Gram-Negative Bacteria*, 16 Current Op. Microbiology 779, 779 (2013), filed as Ex. A, Tab 19 (ECF No. 45-9) (“Zhang”). In contrast, gram-negative bacteria have a flimsy capsule that is surrounded by an outer membrane. Zhang at 779.

possible for a vaccine to cause GBS when its wild antigenic contents had never been so associated. First Whitton Rep. at 11. This was especially troubling, given the distinction between Gram-positive and negative bacteria that Dr. Whitton had observed (which explained why a different bacterial infection was associated with GBS). Second, Dr. Whitton maintained that Dr. Steinman overemphasized studies that had observed the presence of antibodies to phospholipids in the blood of GBS patients, noting that the existence of such antibodies could simply be “the result of the disease,” as the authors of the literature at issue openly admitted. *Id.*; *citing* Nakos, Gilburd. And he disputed the relevance of studies specific to MS, a distinguishable neuropathic illness occurring in the central nervous system, and which had likely different targets for cross-reactive autoimmune attacks. First Whitton Rep. at 11–12.

Next, Dr. Whitton took issue with Dr. Steinman’s contention that polar head groups (which Dr. Whitton defined as “small molecular components”) could be an autoimmune target in GBS—in addition to gangliosides. First Whitton Rep. at 12–13. Overall, he deemed the concept speculative—especially in comparison to how much more was known by medical science about gangliosides. *Id.* at 12. Phospholipids with polar head groups were in fact structurally distinguishable from gangliosides, using a diagram comparing the polar head group phosphocholine with a ganglioside to visually illustrate their differences. *Id.* at 12–13, 16. Thus, even if the pneumococcal vaccine could induce antibodies specific to polar head groups of the kind identified by Dr. Steinman, these antibodies could not *themselves* plausibly cross-react with myelin gangliosides. *Id.* at 13.

In addition, Dr. Whitton characterized as speculative and unsupported Dr. Steinman’s argument that glycerol phosphate was integral to the pneumococcal vaccine’s function (and hence made it more likely that the vaccine would induce antibodies against it). First Whitton Rep. at 13–14. Indeed, Dr. Whitton maintained that strain 10A of the pneumococcal bacterium identified in Dr. Steinman’s report in fact did *not* contain glycerophosphate. *Id.* at 14. Nor did Dr. Whitton accept Dr. Steinman’s contention that phosphorylchlorine had a stimulative/booster effect if part of a vaccine, noting that his reading of the literature offered to support this aspect of Dr. Steinman’s theory did not in fact establish this to be the case. *Id.*

Second Report

Dr. Whitton’s next report provided a point-by-point reaction to Dr. Steinman’s rebuttal efforts. First, he reiterated his prior argument that because MS and GBS are distinguishable, “one should not be used as a model for the other,” and therefore studies specific to MS were not relevant in this case. Second Whitton Rep. at 1. He denied that the two diseases could be lumped together as having a common pathogenesis, and noted that no literature support for Dr. Steinman’s argument had been provided.

Second, Dr. Whitton revisited his general contention that *S. pneumoniae* is not thought by prevailing medical science to be associated with GBS (unlike numerous other bacterial or viral infections). Second Whitton Rep. at 1–2. All Dr. Steinman had offered for an association, he noted, were two case reports (including El Khatib), even though they only established a *temporal* link. *Id.* at 2–3 (discussing case report evidence in detail). Dr. Whitton also provided some additional literature further demonstrating that a lengthy list of GBS bacterial or viral triggers did not include pneumococcus. *See, e.g.,* R. Hughes et al., *Guillain-Barré Syndrome in the 100 Years Since its Description by Guillain, Barré and Strohl*, 139 *Brain* 3041, 3044 (2016), filed as Ex. G, Tab 1 (ECF No. 61-2) (“Hughes”) (“[b]esides *Campylobacter* and Zika virus, the other principal infections established as occurring before GBS more often than chance are cytomegalovirus, *Mycoplasma pneumoniae*,²³ Epstein-Barr virus, *Haemophilus influenzae* and hepatitis E”).

Finally, Dr. Whitton elaborated on his prior argument that phosphoglycerol and polar head groups in nerve myelin were not likely GBS targets, addressing Dr. Steinman’s effort to bulwark that concept but still deeming the overall contention to be speculative. Second Whitton Rep. at 3–4. Dr. Whitton noted that (a) not all of the vaccine’s serotypes featured phosphoglycerol-containing polysaccharides in the first place, (b) the same polysaccharides were contained in the wild bacteria (thus again raising the question why the wild *S. pneumoniae* was not associated with GBS if its counterpart vaccine could), and (c) reliable literature discussing GBS, such as Hughes, said nothing about polar heads groups as a putative target for an autoimmune attack, but repeatedly mentioned gangliosides. *Id.* at 4; *see also* J. Kwan & S. Biliciler, *Guillain-Barré Syndrome and Other Acute Polyneuropathies*, 37 *Clinics Geriatric Med.* 313 (2021), filed as Ex. G, Tab 3 (ECF No. 61-4) (“Kwan”), at 318 (“[i]t is postulated that the epitopes on the surface of pathogens mimic components of the *peripheral nerve ganglioside*, triggering an aberrant activation of the immune system”) (emphasis added). Dr. Whitton also questioned the strength of Dr. Steinman’s argument that the pneumococcal strain that did contain phosphoglycerol (18C), was significant for conjugation—for if so, its chemical structure would be altered and the immunogenicity of the strain effectively destroyed. Second Whitton Rep. at 4.

Third Report

The last written report prepared by Dr. Whitton briefly addressed some of Dr. Steinman’s reiterated prior arguments and his responses (which require no further repetition herein), but he also discussed new issues raised in the third Steinman report, as well.

Dr. Whitton spent some time reacting to the contention (first set forth in Dr. Steinman’s third report) that the pneumococcal vaccine’s *conjugate component* could also trigger antibodies cross-reactive against self mimics, and thereby drive GBS. Third Whitton Rep. at 2–3, 6–8. He

²³ This is not the same as *S. pneumoniae*.

pointed out that in the first two reports filed in this case Dr. Steinman had proposed, without evidence, that phosphoglycerols were a vital link in the attachment of the conjugate to the vaccine's polysaccharide serotypes. Third Whitton Rep. at 6–7, referencing First Steinman Rep. at 12 and Second Steinman Rep. at 13 (suggesting Dr. Steinman's view that phosphorylcholine is itself attached to the conjugate, rather than the polysaccharide serotypes). This assertion, Dr. Whitton maintained, was erroneous. In fact, (a) this “attachment” concept did not account for the serotypes not shown to contain phosphoglycerol, but to which the conjugate also managed to attach, and (b) Dr. Steinman had not explained how (given Chang's findings) the phosphoglycerol so important to the vaccine's immunogenicity would not be altered (thereby eliminating its allegedly-foundational immunogenicity) if it served as a link to the protein conjugate. *Id.* at 7.

Dr. Whitton then engaged in an extended criticism of Dr. Steinman's use of BLAST search data²⁴ to demonstrate the potentiality of mimicry between components of the CRM₁₉₇ conjugate and Contactin-1. Third Whitton Rep. at 8–14. He maintained that the BLAST database was arguably being misused by Dr. Steinman “to try to identify possibly-immunologically-significant tracts of protein,” when the database served a more general purpose, and that the short homologous sequences that Dr. Steinman looked for in his case-specific research were not deserving of “immunological weight.” *Id.* at 10–13. He also argued that the common amino acid sequences shared between Contactin-1 and CRM₁₉₇ that Dr. Steinman had identified were “cherry picked,” with Dr. Steinman focusing only on the results that supported his contentions while ignoring the unhelpful results. *Id.* at 13.

Ultimately, Dr. Whitton concluded that no evidence beyond the scientifically-unreliable homologies revealed via Dr. Steinman's BLAST searches supported the possibility of a cross-reaction with the conjugate. Dr. Steinman had pointed to Raju as identifying a potentially cross-reactive amino acid sequence, but that article did not in fact establish that antibodies generated against the CRM₁₉₇ protein would likely cross-react with Contactin-1. Instead, Raju had focused on the generation of T helper cells in reaction to the actual diphtheria toxin (which is distinguishable from the conjugate, even if the latter synthetically simulates features of the former). Third Whitton Rep. at 17; Raju at 3210. The very sequence at issue was not even one of the peptide sequences that Raju's authors noted *had* been recognized by the studied subject immune responses, whereas “other regions of the diphtheria toxin” were more prominent in stimulating a response. Third Whitton Rep. at 17. Otherwise, the “vast majority” of homologies could not be shown by medical science to always, or likely, result in autoimmune cross-reactive pathologic processes. *Id.* at 14–15 (discussing, for sake of argument, homologies demonstrable between influenza A protein

²⁴ Dr. Whitton similarly criticized Dr. Steinman's “filter funnel” framework for how he reasoned his way to the conclusion that the homologies he demonstrated via BLAST searches reliably established a likely cross-reaction. Third Whitton Rep. at 15–17. He deemed this analytic approach to not only include a number of faulty stages (such as the very effort of looking for homologous sequences), but ultimately reflected circular logic, allowing Dr. Steinman to reach in any case the result most favorable to a petitioner. *Id.* at 17.

sequences in a common vaccine and body show large numbers of common sequences without evidence of actual disease occurring).

Besides the foregoing, Dr. Whitton again addressed the portion of Dr. Steinman's opinion that proposed vaccine-induced antibodies could cross-react with neuronal polar head groups found in nerve myelin. He again deemed the theory speculative and lacking in reliable scientific/medical support, and denied that the vaccine even *required* an immune response to phosphoglycerol specifically for it to function. Third Whitton Rep. at 3. On the contrary, Dr. Whitton maintained that "[t]he role of the phosphoglycerol in bacterial virulence is not relevant" in this context, and that articles like Chang did not establish the existence of an immune response specific to phosphoglycerol, even if its inclusion was important to the vaccine's effectiveness otherwise. *Id.* at 4; Chang at 7091.

Dr. Whitton also commented on Bryson,²⁵ which Dr. Steinman had contended proved an immune response to the vaccine's 23F serotype specific to phosphoglycerol. Third Whitton Rep. at 4–5. He noted that Bryson's authors had actually observed that the immune response to this serotype was specific to "L rhamnose," a large molecule "of which the phosphoglycerol is just one part." *Id.* at 4; Bryson at 4727–28. The targeting observed in Bryson was thus not specific to the smaller phosphoglycerol moiety—and if it were, not only would the immune response to this serotype be sufficient to protect against *other* serotypes (completely contrary to the understanding that the vaccine needed *multiple* serotypes to confer protection, since immune responses were so specific to individual strains), but also (and again), then the wild pneumococcal 23F strain should similarly be capable of causing GBS (yet no reliable evidence suggested this to be the case). *Id.* at 5. In fact, 23F was not even *contained* in Prevnar-13 (and it was speculative for Dr. Steinman to assume comparable phosphoglycerol structures were found in other strains that *are* in the vaccine). *Id.*

²⁵ Independent of Dr. Whitton's criticism, Bryson in fact says even less about causation herein than Dr. Steinman proposes. Bryson focused not on the immunogenicity of the pneumococcal vaccine, how antibodies with affinity for phospholipid components might cause/contribute to GBS, or any other issue directly relevant to this case. Rather, its authors clearly indicated that their study's focus was the role of *preferred genes responsible for coding antibodies to certain pathogens*. Bryson at 4724. Thus, Bryson considered two particular antibodies—one formed in response to the 23F antigenic serotype, and one formed in reaction to the wild cytomegalovirus—choosing them specifically because *both* rely on a "pair of . . . preferred genes" for their generation, even though the two pathogenic incitements are distinguishable. *Id.* at 4723–24. Its authors subsequently looked at the specific structures of the antibodies, finding that they "provide structural evidence that the same . . . genes . . . can assume different roles in protective [antibodies]" due to distinct configurations—and in turn establishing that "evolutionary pressure by pathogens to retain certain . . . genes can collaborate with two stochastic processes." *Id.* at 4724. Bryson's authors concluded that their finding "illustrates how a limited set of human germline genes can contribute to the creation of binding suites for a variety of highly diverse [antigens]." *Id.* at 4729. Bryson seems to have only been cited by Dr. Steinman because it featured a chart showing that the 23F serotype includes phosphoglycerol, and he wished to reproduce it in his written reports.

III. Procedural History

As noted, the Petition in this matter was filed five years ago. Pet. at 1. After some delay attributable to records-gathering efforts, Respondent filed a Rule 4(c) Report in March 2020 contesting Petitioner’s right to compensation. ECF No. 34. Expert reports were filed thereafter, and then the matter was transferred to me in January 2021. Additional reports were filed over the ensuing year, and I subsequently set a schedule for briefing of a ruling on the record in the winter of 2022, along with a date for oral argument on the competing motions. Docket entry, dated February 17, 2022. After some extensions of time, briefing was completed, the oral argument was held in February 2023, and the matter is now fully ripe for resolution.

IV. Parties’ Briefs and Oral Argument

A. Briefing

Petitioner maintains that he has met his causation-in-fact burden based on the factors established by the Federal Circuit in *Althen v. Sec’y of Health & Hum. Servs.*, 418 F.3d 1274 (Fed. Cir. 2005); Mot. at 32–51, 56–60; Reply at 17–32. While much of his briefing focuses on the disputed diagnosis or the other two *Althen* prongs, he also discussed in detail what he believes supports his prong one, “can cause” showing. Since that is the basis for my decision dismissing the claim, I will only summarize those subsections of his briefing, as well as Respondent’s countervailing brief—although Petitioner made a number of very specific arguments pertaining to that aspect of his claim.

Petitioner’s Motion

Petitioner breaks down his causation theory into two parts: one in which the pneumococcal vaccine sparks a cross-reaction as a result of antibodies produced in reaction to phosphoglycerol/phosphocholine found in at least one of the vaccine’s polysaccharide serotypes, and one in which the conjugate itself is the basis for the purported cross-reaction. Mot. at 32, 39. With respect to the former, Petitioner relies heavily on articles submitted by Dr. Steinman about MS, and what they show with respect to phosphate groups on the myelin sheath targeted by autoantibodies, reasoning that they are useful in the GBS context, as well. *Id.* at 33–34, *citing* Ho. He then notes that some of the vaccine’s serotypes have been shown not only to contain these phospholipid-oriented molecules, but that they are critical to immunogenicity. Mot. at 35–37, *citing* Chuang, Chang. Finally, he maintains that articles like Bryson demonstrate that antibodies are generated in response to the phosphoglycerol molecules in at least one of the vaccine’s 13 serotypes. Thus, he contends that via the widely-accepted biologic mechanism of molecular mimicry, these subcomponents of the vaccine trigger the production of autoantibodies specific to polar head structures in the myelin, thereby mistakenly attacking them, relying both on what is

generally known about molecular mimicry in the context of GBS and other wild infections, like *C. jejuni*, and cases (discussed below) where this theory has been accepted. Mot. at 37–39.

Petitioner spends comparatively less time discussing the aspect of his theory relying on the vaccine conjugate. He explains, however, its essence: that Dr. Steinman’s database research had established two possible amino acid sequences that were shared by the conjugate and Contactin-1, a protein found in myelin, and that the lengths of these sequences were sufficient for a cross-reaction to occur. Mot. at 40–44. But this aspect of Dr. Steinman’s opinion does not possess anywhere near the same degree of research suggesting any actual evidence of the theorized cross-reactivity (other than what is found in articles like Lanz—which are specific to MS and the Epstein Barr virus), and Petitioner’s brief does not cite any items of literature to this specific end.

In addition, Petitioner’s brief asserts that certain items of literature actually reveal medical community support for a pneumococcal vaccine-GBS association—although the actual articles make no such affirmative finding, and the readings given them by Petitioner are somewhat strained. Mot. at 56–60. Haber, for example, was deemed by Dr. Steinman as at least “reassuring” in its determination that the vaccine is not associated with a disproportionate number of GBS cases, but he maintained (especially since it relies on VAERS data) that it could not disprove an association. *Id.* at 57. Tseng did not compare its findings regarding vaccinated individuals who experienced GBS against a background incidence rate, while seeing some instances of GBS regardless. *Id.* at 58–59. And Baxter did not involve the relevant vaccine, and had other limitations. *Id.* at 60.

Respondent’s Brief

Respondent denies that the first *Althen* prong has been satisfied. Opp. at 18–23.²⁶ The fact that the underlying *S. pneumoniae* bacterium is not associated with GBS is a fatal flaw to the theory, and nothing offered specific to it could overcome this foundational problem. *Id.* at 18–19. This might be because the kinds of molecules found in other bacteria that *are* so associated, like *C. jejuni*, express a different kind of molecule *known* to mimic aspects of myelin. *Id.* at 19. And Petitioner did not offer evidence associating the vaccine with GBS, while the existing epidemiology undercuts an association. *Id.* at 19–20.

The theory offered herein otherwise relies on unsubstantiated contentions about molecular mimicry that have been rejected in prior cases also involving the pneumococcal vaccine (as

²⁶ Respondent also makes some arguments about the adequacy of Dr. Steinman’s expert opinion, given his propensity in prior cases (which I have observed myself) to act more as an advocate than independent expert. Opp. at 7–8. But my determination herein relies on the “face value” of Dr. Steinman’s opinion (albeit weighed against my prior exposure to it) and thus I do not comment on his individual proclivities in Program cases generally (beyond how needlessly long and diagram-laden his reports tend to be).

discussed below). Opp. at 20. In particular, it assumes without preponderant evidence that (a) phospholipid polar head groups can be a target of GBS much like ganglioside structures (even though substantially more science supports the latter, with hardly any going the other way), and (b) antibodies to these polar head groups can drive disease, simply based on the fact that in a few studies they have been found in the blood of GBS patients. *Id.* at 21, *citing* Nakos, Gilburd. Dr. Steinman also maintained that phosphoglycerols were vital to the vaccine’s immunogenicity—when in fact, as Dr. Whitton established, Petitioner’s own literature suggested that they would not exist after the vaccine’s manufacture (which involves connection of all included serotypes to the protein conjugate) if they functioned as proposed. *Id.* at 21–22, *citing* Chang.

Respondent also criticized Dr. Steinman’s BLAST searches, which were specific to the conjugate “side” of his theory, as not probative of causation. Opp. at 22. Not only did Dr. Steinman not show that Contactin-1 is a likely GBS-instigating target, but he also demonstrated insufficient numbers of amino acid sequences to trigger a cross-reaction, as explained by Dr. Whitton. *Id.* And he relied on case reports that were factually distinguishable, like El Khatib. *Id.* at 23.

Reply

Only a portion of Petitioner’s reply focuses on his prong one showing, and it mostly reiterates the arguments addressed above. Reply at 17–23. Petitioner does, however, propose that evidence offered in the case shows that gangliosides are not the only target in GBS, and that autoantibodies to phospholipids are seen in connection with GBS patients in some studies. *Id.* at 18–19, *citing* Nakos. The evidence favoring gangliosides as a target, Petitioner maintained, is only specific to *C. jejuni* infections, and thus does not suggest that “different immune challenges” could not involve different aspects of the myelin. Reply at 19. (And in any event, the record evidence does not establish that Petitioner tested positive for anti-ganglioside antibodies). *Id.*

Petitioner also maintained that even if articles like Nakos expressly decline to say whether anti-phospholipid antibodies are causal of GBS (as opposed to the indirect product of the myelin damage associated with GBS), other articles do support the conclusion—albeit in the distinguishable context of MS. Reply at 20, *citing* Ho. He also notes the numerous other decisions accepting Dr. Steinman’s reasoning, deeming them the equivalent of case reports. Reply at 20–21 (*citations omitted*). And he again walks through Dr. Steinman’s BLAST search-supported theory involving the vaccine’s CRM₁₉₇ component. *Id.* at 22–23.

B. Oral Argument

The parties agreed to have their respective counsel present live argument in court, rather than conduct a hearing featuring expert testimony, and to that end I heard both sides in February 2023. At that time, Petitioner maintained he had successfully established all elements of his claim,

but in so maintaining offered substantial commentary on the *Althen* prong one issues I am focusing upon in this Decision. *See generally* Tr. at 7–19, 25–47, 66–71. The argument lasted two hours.

In particular, Petitioner’s counsel reiterated the elements and components of Dr. Steinman’s causation theory, noting its two main alternative mechanistic contentions (cross-reaction to phosphoglycerol moieties, plus cross-reaction against the conjugate)²⁷ and also highlighting literature already discussed above. Tr. at 7–12 (mentioning Bryson, Chang, Nakos, and Ho, among other items). He proposed that the theory offered was comparable to what is known about the *C. jejuni*—GBS relationship. *Id.* at 12.²⁸ And he noted that several prior special masters had ruled favorably on the theory, whereas the sole decision that I had issued at the time of hearing stood as an outlier, and also did not involve a theory presented by Dr. Steinman.²⁹ *Id.* at 15, 17–18.

I also asked questions of counsel about how the preponderance standard should be applied *generally* in causation cases—and in effect, although counsel agreed the standard of proof *was* preponderance, he proposed that to deny that Petitioner had met it herein on the first prong would be to improperly heighten his burden. Thus, counsel maintained that questions about whether anti-phosphoglycerol antibodies likely cause GBS (as opposed to appearing merely in the *context* of the disease)³⁰—questions that literature filed by Petitioner expressly *do not propose to answer*—amounted to “the ‘when’ and the ‘what’” questions that “may be necessary for scientific certainty but not for a preponderance of the evidence or legal probability.” Tr. at 16. Thus, as long as a claimant has offered individually-reliable items of literature that are not shown to be false or scientifically-untrustworthy, plus a qualified expert (like Dr. Steinman) embracing the theory, “we meet *Althen* 1.” *Id.* at 35, 38. Any weighing performed by the special master should thus be limited to either evaluating if in fact the items offered are as individually reliable as they are held out to be, whether they have been rebutted by counter-evidence offered by Respondent that contradicts their findings (such as additional studies on the same topic), or whether other kinds of evidence

²⁷ Counsel did acknowledge indirectly that the first theoretical contention was “the stronger theory,” although Petitioner has not abandoned the aspect of the theory relying on the conjugate. Tr. at 6.

²⁸ Counsel acknowledged, however, that the evidence of an association between the underlying wild *S. pneumoniae* bacteria and GBS was wanting (or at least far less robust than associations known with other wild viruses or bacteria, such as *C. jejuni*). Tr. at 27–28, 69. But he later stressed that vaccine’s formulation as a conjugated vaccine might explain why the vaccine would more likely spark an autoimmune disease than the wild bacteria. *Id.* at 67–68.

²⁹ At the time of oral argument, my third-published pneumococcal vaccine-GBS decision (which specifically discussed Dr. Steinman’s theory as offered herein) had not yet been issued—but was the following week. *See Trollinger*, 2023 WL 2521912, at *4–6.

³⁰ By “context,” I mean that the antibodies might be generated mid-disease course—not from driving the disease initially, and/or arising as the body reacts to the damage from the disease rather than increasing the disease tempo or worsening it. Certainly, Petitioner’s literature filings say nothing about the likelihood that the antibodies drive initiating the autoimmune process leading to GBS.

(such as a particularly persuasive epidemiologic study—evidence I acknowledge does not exist in this case) merit greater weight.

Respondent offered his own argument in favor of dismissal. Tr. at 48–66. His counsel contended that my prior decisions provided a reasoned roadmap for resolving this case, especially since nothing different had actually been proposed as grounds for finding entitlement in this record, and that Petitioner was in effect relying on the flu vaccine-GBS theory but in a disparate context. *Id.* at 50–51. Dr. Steinman’s theory was in fact not reliable, for the reasons discussed above in my summary of Dr. Whitton’s opinion. *Id.* at 51–52, 57–59. Respondent thus maintained that the collective weight of Petitioner’s evidence offered in support of the theory was not preponderantly sufficient to meet the “can cause” prong standard. In Respondent’s view, “where” or “when” evidence specific to the putative role of anti-phosphoglycerol antibodies was lacking, and it did not amount to an unreasonable heightening of the preponderant burden to require such evidence. *Id.* at 64–65 (“they need to have evidence to show that the autoantibodies would be pathogenic”).

V. Applicable Law

A. *Standards for Vaccine Claims*

To receive compensation in the Vaccine Program, a petitioner must prove either: (1) that he suffered a “Table Injury”—i.e., an injury falling within the Vaccine Injury Table—corresponding to one of the vaccinations in question within a statutorily prescribed period of time or, in the alternative, (2) that his illnesses were actually caused by a vaccine (a “Non-Table Injury”). See Sections 13(a)(1)(A), 11(c)(1), and 14(a), as amended by 42 C.F.R. § 100.3; § 11(c)(1)(C)(ii)(I); see also *Moberly v. Sec’y of Health & Hum. Servs.*, 592 F.3d 1315, 1321 (Fed. Cir. 2010); *Capizzano v. Sec’y of Health & Hum. Servs.*, 440 F.3d 1317, 1320 (Fed. Cir. 2006).³¹ In this case, Petitioner cannot assert a Table claim (as *there is no such claim* with respect to the pneumococcal vaccine).

For both Table and Non-Table claims, Vaccine Program petitioners bear a “preponderance of the evidence” burden of proof. Section 13(1)(a). That is, a petitioner must offer evidence that leads the “trier of fact to believe that the existence of a fact is more probable than its nonexistence before [he] may find in favor of the party who has the burden to persuade the judge of the fact’s existence.” *Moberly*, 592 F.3d at 1322 n.2; see also *Snowbank Enter. v. United States*, 6 Cl. Ct. 476, 486 (1984) (mere conjecture or speculation is insufficient under a preponderance standard). Proof of medical certainty is not required. *Bunting v. Sec’y of Health & Hum. Servs.*, 931 F.2d 867, 873 (Fed. Cir. 1991). In particular, a petitioner must demonstrate that the vaccine was “not

³¹ Decisions of special masters (some of which I reference in this ruling) constitute persuasive but not binding authority. *Hanlon v. Sec’y of Health & Hum. Servs.*, 40 Fed. Cl. 625, 630 (1998). By contrast, Federal Circuit rulings concerning legal issues are binding on special masters. *Guillory v. Sec’y of Health & Hum. Servs.*, 59 Fed. Cl. 121, 124 (2003), *aff’d* 104 F. Appx. 712 (Fed. Cir. 2004); see also *Spooner v. Sec’y of Health & Hum. Servs.*, No. 13-159V, 2014 WL 504728, at *7 n.12 (Fed. Cl. Spec. Mstr. Jan. 16, 2014).

only [the] but-for cause of the injury but also a substantial factor in bringing about the injury.” *Moberly*, 592 F.3d at 1321 (quoting *Shyface v. Sec’y of Health & Hum. Servs.*, 165 F.3d 1344, 1352–53 (Fed. Cir. 1999)); *Pafford v. Sec’y of Health & Hum. Servs.*, 451 F.3d 1352, 1355 (Fed. Cir. 2006). A petitioner may not receive a Vaccine Program award based solely on his assertions; rather, the petition must be supported by either medical records or by the opinion of a competent physician. Section 13(a)(1).

In attempting to establish entitlement to a Vaccine Program award of compensation for a Non-Table claim, a petitioner must satisfy all three of the elements established by the Federal Circuit in *Althen*, 418 F.3d at 1278: “(1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of proximate temporal relationship between vaccination and injury.”

Each of the *Althen* prongs requires a different showing. Under *Althen* prong one, petitioners must provide a “reputable medical theory,” demonstrating that the vaccine received *can cause* the type of injury alleged. *Pafford*, 451 F.3d at 1355–56 (citations omitted). To satisfy this prong, a petitioner’s theory must be based on a “sound and reliable medical or scientific explanation.” *Knudsen v. Sec’y of Health & Hum. Servs.*, 35 F.3d 543, 548 (Fed. Cir. 1994). Such a theory must only be “legally probable, not medically or scientifically certain.” *Knudsen*, 35 F.3d at 549.

Petitioners may satisfy the first *Althen* prong without resort to medical literature, epidemiological studies, demonstration of a specific mechanism, or a generally accepted medical theory. *Andreu v. Sec’y of Health & Hum. Servs.*, 569 F.3d 1367, 1378–79 (Fed. Cir. 2009) (citing *Capizzano*, 440 F.3d at 1325–26). Special masters, despite their expertise, are not empowered by statute to conclusively resolve what are essentially thorny scientific and medical questions, and thus scientific evidence offered to establish *Althen* prong one is viewed “not through the lens of the laboratorian, but instead from the vantage point of the Vaccine Act’s preponderant evidence standard.” *Id.* at 1380. Accordingly, special masters must take care not to increase the burden placed on petitioners in offering a scientific theory linking vaccine to injury. *Contreras*, 121 Fed. Cl. at 245.

In discussing the evidentiary standard applicable to the first *Althen* prong, the Federal Circuit has consistently rejected the contention that it can be satisfied merely by establishing the proposed causal theory’s scientific or medical *plausibility*. See *Boatmon v. Sec’y of Health & Hum. Servs.*, 941 F.3d 1351, 1359 (Fed. Cir. 2019); *LaLonde v. Sec’y of Health & Hum. Servs.*, 746 F.3d 1334, 1339 (Fed. Cir. 2014) (“[h]owever, in the past we have made clear that simply identifying a ‘plausible’ theory of causation is insufficient for a petitioner to meet her burden of proof” (citing *Moberly*, 592 F.3d at 1322)); see also *Howard v. Sec’y of Health & Hum. Servs.*, 2023 WL 4117370, at *4 (Fed. Cl. May 18, 2023) (“[t]he standard has been preponderance for nearly four

decades”), *appeal docketed*, No. 23-1816 (Fed. Cir. Apr. 28, 2023). And petitioners always have the ultimate burden of establishing their *overall* Vaccine Act claim with preponderant evidence. *W.C. v. Sec’y of Health & Hum. Servs.*, 704 F.3d 1352, 1356 (Fed. Cir. 2013) (citations omitted); *Tarsell v. United States*, 133 Fed. Cl. 782, 793 (2017) (noting that *Moberly* “addresses the petitioner’s overall burden of proving causation-in-fact under the Vaccine Act” by a preponderance standard).

The second *Althen* prong requires proof of a logical sequence of cause and effect, usually supported by facts derived from a petitioner’s medical records. *Althen*, 418 F.3d at 1278; *Andreu*, 569 F.3d at 1375–77; *Capizzano*, 440 F.3d at 1326; *Grant v. Sec’y of Health & Hum. Servs.*, 956 F.2d 1144, 1148 (Fed. Cir. 1992). In establishing that a vaccine “did cause” injury, the opinions and views of the injured party’s treating physicians are entitled to some weight. *Andreu*, 569 F.3d at 1367; *Capizzano*, 440 F.3d at 1326 (“medical records and medical opinion testimony are favored in vaccine cases, as treating physicians are likely to be in the best position to determine whether a ‘logical sequence of cause and effect show[s] that the vaccination was the reason for the injury’”) (quoting *Althen*, 418 F.3d at 1280). Medical records are generally viewed as particularly trustworthy evidence, since they are created contemporaneously with the treatment of the patient. *Cucuras v. Sec’y of Health & Hum. Servs.*, 993 F.2d 1525, 1528 (Fed. Cir. 1993).

Medical records and statements of a treating physician, however, do not *per se* bind the special master to adopt the conclusions of such an individual, even if they must be considered and carefully evaluated. Section 13(b)(1) (providing that “[a]ny such diagnosis, conclusion, judgment, test result, report, or summary shall not be binding on the special master or court”); *Snyder v. Sec’y of Health & Hum. Servs.*, 88 Fed. Cl. 706, 746 n.67 (2009) (“there is nothing . . . that mandates that the testimony of a treating physician is sacrosanct—that it must be accepted in its entirety and cannot be rebutted”). As with expert testimony offered to establish a theory of causation, the opinions or diagnoses of treating physicians are only as trustworthy as the reasonableness of their suppositions or bases. The views of treating physicians should be weighed against other, contrary evidence also present in the record—including conflicting opinions among such individuals. *Hibbard v. Sec’y of Health & Hum. Servs.*, 100 Fed. Cl. 742, 749 (2011) (not arbitrary or capricious for special master to weigh competing treating physicians’ conclusions against each other), *aff’d*, 698 F.3d 1355 (Fed. Cir. 2012); *Veryzer v. Sec’y of Dept. of Health & Hum. Servs.*, No. 06-522V, 2011 WL 1935813, at *17 (Fed. Cl. Spec. Mstr. Apr. 29, 2011), *mot. for review denied*, 100 Fed. Cl. 344, 356 (2011), *aff’d without opinion*, 475 F. Appx. 765 (Fed. Cir. 2012).

The third *Althen* prong requires establishing a “proximate temporal relationship” between the vaccination and the injury alleged. *Althen*, 418 F.3d at 1281. That term has been equated to the phrase “medically-acceptable temporal relationship.” *Id.* A petitioner must offer “preponderant proof that the onset of symptoms occurred within a timeframe which, given the medical understanding of the disorder’s etiology, it is medically acceptable to infer causation.” *de Bazan*

v. Sec’y of Health & Hum. Servs., 539 F.3d 1347, 1352 (Fed. Cir. 2008). The explanation for what is a medically acceptable timeframe must align with the theory of how the relevant vaccine can cause an injury (*Althen* prong one’s requirement). *Id.* at 1352; *Shapiro v. Sec’y of Health & Hum. Servs.*, 101 Fed. Cl. 532, 542 (2011), *recons. denied after remand*, 105 Fed. Cl. 353 (2012), *aff’d mem.*, 503 F. Appx. 952 (Fed. Cir. 2013); *Koehn v. Sec’y of Health & Hum. Servs.*, No. 11-355V, 2013 WL 3214877 (Fed. Cl. Spec. Mstr. May 30, 2013), *mot. for review denied*, (Fed. Cl. Dec. 3, 2013), *aff’d*, 773 F.3d 1239 (Fed. Cir. 2014).

B. Law Governing Analysis of Fact Evidence

The process for making determinations in Vaccine Program cases regarding factual issues begins with consideration of the medical records. Section 11(c)(2). The special master is required to consider “all [] relevant medical and scientific evidence contained in the record,” including “any diagnosis, conclusion, medical judgment, or autopsy or coroner’s report which is contained in the record regarding the nature, causation, and aggravation of the petitioner’s illness, disability, injury, condition, or death,” as well as the “results of any diagnostic or evaluative test which are contained in the record and the summaries and conclusions.” Section 13(b)(1)(A). The special master is then required to weigh the evidence presented, including contemporaneous medical records and testimony. *See Burns v. Sec’y of Health & Hum. Servs.*, 3 F.3d 415, 417 (Fed. Cir. 1993) (determining that it is within the special master’s discretion to determine whether to afford greater weight to contemporaneous medical records than to other evidence, such as oral testimony surrounding the events in question that was given at a later date, provided that such determination is evidenced by a rational determination).

As noted by the Federal Circuit, “[m]edical records, in general, warrant consideration as trustworthy evidence.” *Cucuras*, 993 F.2d at 1528; *Doe/70 v. Sec’y of Health & Hum. Servs.*, 95 Fed. Cl. 598, 608 (2010) (“[g]iven the inconsistencies between petitioner’s testimony and his contemporaneous medical records, the special master’s decision to rely on petitioner’s medical records was rational and consistent with applicable law”), *aff’d*, *Rickett v. Sec’y of Health & Hum. Servs.*, 468 F. App’x 952 (Fed. Cir. 2011) (non-precedential opinion). A series of linked propositions explains why such records deserve some weight: (i) sick people visit medical professionals; (ii) sick people attempt to honestly report their health problems to those professionals; and (iii) medical professionals record what they are told or observe when examining their patients in as accurate a manner as possible, so that they are aware of enough relevant facts to make appropriate treatment decisions. *Sanchez v. Sec’y of Health & Hum. Servs.*, No. 11–685V, 2013 WL 1880825, at *2 (Fed. Cl. Spec. Mstr. Apr. 10, 2013); *Cucuras v. Sec’y of Health & Hum. Servs.*, 26 Cl. Ct. 537, 543 (1992), *aff’d*, 993 F.2d 1525 (Fed. Cir. 1993) (“[i]t strains reason to conclude that petitioners would fail to accurately report the onset of their daughter’s symptoms”).

Accordingly, if the medical records are clear, consistent, and complete, then they should be afforded substantial weight. *Lowrie v. Sec’y of Health & Hum. Servs.*, No. 03–1585V, 2005 WL

6117475, at *20 (Fed. Cl. Spec. Mstr. Dec. 12, 2005). Indeed, contemporaneous medical records are often found to be deserving of greater evidentiary weight than oral testimony—especially where such testimony conflicts with the record evidence. *Cucuras*, 993 F.2d at 1528; *see also* *Murphy v. Sec’y of Health & Hum. Servs.*, 23 Cl. Ct. 726, 733 (1991), *aff’d per curiam*, 968 F.2d 1226 (Fed. Cir. 1992), *cert. denied*, *Murphy v. Sullivan*, 506 U.S. 974 (1992) (citing *United States v. United States Gypsum Co.*, 333 U.S. 364, 396 (1947) (“[i]t has generally been held that oral testimony which is in conflict with contemporaneous documents is entitled to little evidentiary weight.”)).

However, the Federal Circuit has also noted that there is no formal “presumption” that records are accurate or superior on their face to other forms of evidence. *Kirby v. Sec’y of Health & Hum. Servs.*, 997 F.3d 1378, 1383 (Fed. Cir. 2021). There are certainly situations in which compelling oral testimony may be more persuasive than written records, such as where records are deemed to be incomplete or inaccurate. *Campbell v. Sec’y of Health & Hum. Servs.*, 69 Fed. Cl. 775, 779 (2006) (“like any norm based upon common sense and experience, this rule should not be treated as an absolute and must yield where the factual predicates for its application are weak or lacking”); *Lowrie*, 2005 WL 6117475, at *19 (“[w]ritten records which are, themselves, inconsistent, should be accorded less deference than those which are internally consistent”) (quoting *Murphy*, 23 Cl. Ct. at 733)). Ultimately, a determination regarding a witness's credibility is needed when determining the weight that such testimony should be afforded. *Andreu*, 569 F.3d at 1379; *Bradley v. Sec’y of Health & Hum. Servs.*, 991 F.2d 1570, 1575 (Fed. Cir. 1993).

When witness testimony is offered to overcome the presumption of accuracy afforded to contemporaneous medical records, such testimony must be “consistent, clear, cogent, and compelling.” *Sanchez*, 2013 WL 1880825, at *3 (citing *Blutstein v. Sec’y of Health & Hum. Servs.*, No. 90–2808V, 1998 WL 408611, at *5 (Fed. Cl. Spec. Mstr. June 30, 1998)). In determining the accuracy and completeness of medical records, the Court of Federal Claims has listed four possible explanations for inconsistencies between contemporaneously created medical records and later testimony: (1) a person's failure to recount to the medical professional everything that happened during the relevant time period; (2) the medical professional's failure to document everything reported to her or him; (3) a person's faulty recollection of the events when presenting testimony; or (4) a person's purposeful recounting of symptoms that did not exist. *La Londe v. Sec’y of Health & Hum. Servs.*, 110 Fed. Cl. 184, 203–04 (2013), *aff’d*, 746 F.3d 1334 (Fed. Cir. 2014). In making a determination regarding whether to afford greater weight to contemporaneous medical records or other evidence, such as testimony at hearing, there must be evidence that this decision was the result of a rational determination. *Burns*, 3 F.3d at 417.

C. *Analysis of Expert Testimony*

Establishing a sound and reliable medical theory often requires a petitioner to present expert testimony in support of his claim. *Lampe v. Sec’y of Health & Hum. Servs.*, 219 F.3d 1357, 1361 (Fed. Cir. 2000). Vaccine Program expert testimony is usually evaluated according to the factors for analyzing scientific reliability set forth in *Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 509 U.S. 579, 594–96 (1993). See *Cedillo v. Sec’y of Health & Hum. Servs.*, 617 F.3d 1328, 1339 (Fed. Cir. 2010) (citing *Terran v. Sec’y of Health & Hum. Servs.*, 195 F.3d 1302, 1316 (Fed. Cir. 1999)). “The *Daubert* factors for analyzing the reliability of testimony are: (1) whether a theory or technique can be (and has been) tested; (2) whether the theory or technique has been subjected to peer review and publication; (3) whether there is a known or potential rate of error and whether there are standards for controlling the error; and (4) whether the theory or technique enjoys general acceptance within a relevant scientific community.” *Terran*, 195 F.3d at 1316 n.2 (citing *Daubert*, 509 U.S. at 592–95).

The *Daubert* factors play a slightly different role in Vaccine Program cases than they do when applied in other federal judicial fora (such as the district courts). *Daubert* factors are usually employed by judges (in the performance of their evidentiary gatekeeper roles) to exclude evidence that is unreliable and/or could confuse a jury. In Vaccine Program cases, by contrast, these factors are used in the *weighing* of the reliability of scientific evidence proffered. *Davis v. Sec’y of Health & Hum. Servs.*, 94 Fed. Cl. 53, 66–67 (2010) (“uniquely in this Circuit, the *Daubert* factors have been employed also as an acceptable evidentiary-gauging tool with respect to persuasiveness of expert testimony already admitted”). The flexible use of the *Daubert* factors to evaluate the persuasiveness and reliability of expert testimony has routinely been upheld. See *e.g.*, *Snyder*, 88 Fed. Cl. at 742–45. In this matter (as in numerous other Vaccine Program cases), *Daubert* has not been employed at the threshold, to determine what evidence should be admitted, but instead to determine whether expert testimony offered is reliable and/or persuasive.

Respondent frequently offers one or more experts of his own in order to rebut a petitioner’s case. Where both sides offer expert testimony, a special master’s decision may be “based on the credibility of the experts and the relative persuasiveness of their competing theories.” *Broekelschen v. Sec’y of Health & Hum. Servs.*, 618 F.3d 1339, 1347 (Fed. Cir. 2010) (citing *Lampe*, 219 F.3d at 1362). However, nothing requires the acceptance of an expert’s conclusion “connected to existing data only by the *ipse dixit* of the expert,” especially if “there is simply too great an analytical gap between the data and the opinion proffered.” *Snyder*, 88 Fed. Cl. at 743 (quoting *Gen. Elec. Co. v. Joiner*, 522 U.S. 136, 146 (1997)); see also *Isaac v. Sec’y of Health & Hum. Servs.*, No. 08-601V, 2012 WL 3609993, at *17 (Fed. Cl. Spec. Mstr. July 30, 2012), *mot. for rev. denied*, 108 Fed. Cl. 743 (2013), *aff’d*, 540 F. Appx. 999 (Fed. Cir. 2013) (citing *Cedillo*, 617 F.3d at 1339). Weighing the relative persuasiveness of competing expert testimony, based on a particular expert’s credibility, is part of the overall reliability analysis to which special masters

must subject expert testimony in Vaccine Program cases. *Moberly*, 592 F.3d at 1325–26 (“[a]ssessments as to the reliability of expert testimony often turn on credibility determinations”); *see also Porter v. Sec’y of Health & Hum. Servs.*, 663 F.3d 1242, 1250 (Fed. Cir. 2011) (“this court has unambiguously explained that special masters are expected to consider the credibility of expert witnesses in evaluating petitions for compensation under the Vaccine Act”).

Expert opinions based on unsupported facts may be given relatively little weight. *See Dobrydney v. Sec’y of Health & Hum. Servs.*, 556 F. Appx. 976, 992–93 (Fed. Cir. 2014) (“[a] doctor’s conclusion is only as good as the facts upon which it is based”) (citing *Brooke Group Ltd. v. Brown & Williamson Tobacco Corp.*, 509 U.S. 209, 242 (1993) (“[w]hen an expert assumes facts that are not supported by a preponderance of the evidence, a finder of fact may properly reject the expert’s opinion”)). Expert opinions that fail to address or are at odds with contemporaneous medical records may therefore be less persuasive than those which correspond to such records. *See Gerami v. Sec’y of Health & Hum. Servs.*, No. 12-442V, 2013 WL 5998109, at *4 (Fed. Cl. Spec. Mstr. Oct. 11, 2013), *aff’d*, 127 Fed. Cl. 299 (2014).

D. *Consideration of Medical Literature*

Both parties filed numerous items of medical and scientific literature in this case, but not every filed item factors into the outcome of this Decision. While I have reviewed all the medical literature submitted in this case, I discuss only those articles that are most relevant to my determination and/or are central to Petitioner’s case—just as I have not exhaustively discussed every individual medical record filed. *Moriarty v. Sec’y of Health & Hum. Servs.*, 844 F.3d 1322, 1328 (Fed. Cir. 2016) (“[w]e generally presume that a special master considered the relevant record evidence even though he does not explicitly reference such evidence in his decision”) (citation omitted); *see also Paterek v. Sec’y of Health & Hum. Servs.*, 527 F. Appx. 875, 884 (Fed. Cir. 2013) (“[f]inding certain information not relevant does not lead to—and likely undermines—the conclusion that it was not considered”).

E. *Standards for Ruling on the Record*

I am resolving Petitioner’s claim on the filed record, in accordance with the parties’ wishes as well as my own assessment of how best to decide the claim. The Vaccine Act and Rules not only contemplate but encourage special masters to decide petitions on the papers where (in the exercise of their discretion) they conclude that doing so will properly and fairly resolve the case. Section 12(d)(2)(D); Vaccine Rule 8(d). The decision to rule on the record in lieu of hearing has been affirmed on appeal. *Kreizenbeck v. Sec’y of Health & Hum. Servs.*, 945 F.3d 1362, 1366 (Fed. Cir. 2020); *see also Hooker v. Sec’y of Health & Hum. Servs.*, No. 02-472V, 2016 WL 3456435, at *21 n.19 (Fed. Cl. Spec. Mstr. May 19, 2016) (citing numerous cases where special masters decided case on the papers in lieu of hearing and that decision was upheld). I am simply not required to hold a hearing in every matter, no matter the preferences of the parties. *Hovey v. Sec’y*

of Health & Hum. Servs., 38 Fed. Cl. 397, 402–03 (1997) (determining that special master acted within his discretion in denying evidentiary hearing); *Burns*, 3 F.3d at 417; *Murphy v. Sec’y of Health & Hum. Servs.*, No. 90-882V, 1991 WL 71500, at *2 (Fed. Cl. Spec. Mstr. Apr. 19, 1991).

ANALYSIS

I. Overview of Prior Decisions

Much is known about GBS’s likely pathogenesis, as well as its association with one particular vaccine covered by the Program: the flu vaccine. There are several reliable evidentiary components supporting this association, as I have discussed in other cases.³² *Trollinger v. Sec’y of Health & Hum. Servs.*, No. 16-473V, 2023 WL 2521912, at *25 (Fed. Cl. Spec. Mstr. Feb. 17, 2023), *mot. for review den’d*, No. 16-VV-473, 2023 WL 5249583 (Fed. Cl. 2023); *Bielak v. Sec’y of Health & Hum. Servs.*, No. 18-761V, 2022 WL 18058244, at *34 (Fed. Cl. Spec. Mstr. Dec. 9, 2022). These evidentiary components have been deemed sufficient (on numerous prior occasions) to preponderantly demonstrate the flu vaccine can likely cause GBS. *Mason v. Sec’y of Health & Hum. Servs.*, No. 17-1383V, 2022 WL 600415, at *26 (Fed. Cl. Spec. Mstr. Feb. 4, 2022) (noting that the flu vaccine-GBS association is supported by a “mix of (a) knowledge about how molecular mimicry “works” in GBS’s pathogenesis, (b) trustworthy animal experiments that model demyelinating injuries in the context of the molecular mimicry mechanism, and (c) solid (if somewhat old) epidemiologic evidence . . . establishing a higher incidence of GBS after vaccination when compared to an unvaccinated population”). As a result, there are many reasoned Program determinations recognizing the strength of the association.³³ Indeed, GBS after the flu vaccine is a Table claim, as well. 42 C.F.R. § 100.3(a)(XIV)(D).

The same is not true for the pneumococcal vaccine, however. On the contrary: to date I have issued three lengthy decisions explaining in detail why it is unlikely that the pneumococcal vaccine can cause GBS. *See generally Trollinger*, 2023 WL 2521912, at *27–30; *Bielak*, 2022 WL

³² Although I decide this case based on the evidence before me, it is not only useful, but prudent, to take into account prior determinations—both for guidance and to avoid “reinventing the wheel” when deciding like Program cases.

³³ *See, e.g., China v. Sec’y of Health & Human Servs.*, No. 15-095V, 2019 WL 1873322 (Fed. Cl. Spec. Mstr. Mar. 15, 2019); *Strong v. Sec’y of Health & Human Servs.*, No. 15-1108V, 2018 WL 1125666 (Fed. Cl. Spec. Mstr. Jan. 12, 2018); *Stitt v. Sec’y of Health & Human Servs.*, No. 09-653V, 2013 WL 3356791 (Fed. Cl. Spec. Mstr. May 31, 2013); *Stewart v. Sec’y of Health & Human Servs.*, No. 06-777V, 2011 WL 3241585, at *16 (Fed. Cl. Spec. Mstr. July 8, 2011). Such cases often also rely on the theory of molecular mimicry, proposing that antibodies produced by B cells in response to a vaccine’s viral antigen components can cross-attack the myelin sheath (because the target antigen and gangliosides of the myelin sheath share structural homology), thereby causing demyelination of peripheral nerves. *China*, 2019 WL 1873322, at *15.

18058244, at *33–37 (pneumococcal vaccine not shown to cause GBS); *Deshler v. Sec'y of Health & Hum. Servs.*, No. 16-1070V, 2020 WL 4593162 (Fed. Cl. Spec. Mstr. July 1, 2020).

Bielak and *Trollinger* involved theories largely comparable to what was offered here, as in both cases the petitioner argued that phosphoglycerol components of the pneumococcal vaccine caused antibodies to cross-react against relevant portions of the myelin, and both also involved nearly all of the same items of literature, like Ho, Gilburd, Nakos, etc. *See Trollinger*, 2023 WL 2521912, at *19; *Bielak*, 2023 WL 35509, at *15–17, 33–34. The *Trollinger* petitioner also featured the expert input of Dr. Steinman, whose report (after an initial, slightly-different iteration) ended up consistent with what has been proposed in this case (and with Dr. Whitton serving the role as Respondent's expert, as well).

My determination in *Deshler*, 2020 WL 4593162, also bears on the outcome herein, if less directly. The *Deshler* petitioner relied on the second component of Dr. Steinman's theory: that the conjugate component of the Prevnar-13 vaccine *itself* had caused an aberrant, mimicry-driven autoimmune process relating to the vaccine's antigens. *Deshler*, 2020 WL 4593162, at *27. That petitioner's expert opined the subsequent B cell reaction (the primary goal of the vaccine) was driven by the polysaccharide component of the vaccination, although (unlike this case) he conceded that he could not demonstrate mimicry between the *S. pneumoniae* polysaccharides and self-structures. *Id.* Respondent's expert in *Deshler* (Dr. Whitton again) argued in reaction that the polysaccharides contained in the vaccine did not share structural homology with self-structures of the peripheral nervous system, and thus could not contribute to the pathogenesis of GBS via a molecular mimicry-driven cross-reaction to the vaccine's antigens. *Id.* at *27. I concurred with Respondent, while also finding that the petitioner relied too heavily on the temporal association between vaccination and onset as evidence of causation (and that there was another potential explanation for the claimant's GBS that had not been rebutted). *Id.* at *22, 27.

It is unquestionably the case (as I previously observed in *Trollinger* and *Bielak*) that other special masters have accepted Dr. Steinman's theory regarding GBS and the pneumococcal vaccine. *See, e.g., Gross v. Sec'y of Health & Hum. Servs.*, No. 17-1075V, 2022 WL 9669651 (Fed. Cl. Spec. Mstr. Sept. 22, 2022); *Maloney*, 2022 WL 1074087; *Pierson v. Sec'y of Health & Hum. Servs.*, No. 17-1136V, 2022 WL 322836 (Fed. Cl. Spec. Mstr. Jan. 19, 2022); *Koller v. Sec'y of Health & Hum. Servs.*, No. 16-439V, 2021 WL 5027947 (Fed. Cl. Spec. Mstr. Oct. 8, 2021). However (and although I am not bound by these determinations in any event), I do not find them persuasive. They largely seem to have embraced the framework for the existing flu vaccine-GBS association, but without convincingly explaining why Respondent's counter-arguments deserved less weight given the disparate context. I have also observed that these favorable cases cite the same items of literature that I determined (after close analysis) are not especially reliable or persuasive. *See, e.g., Bielak*, 2022 WL 18058244, at *15–17, 32 (discussing in detail Chang, Gilburd, Ho, Kanter, and Nakos).

Ultimately, as I explained in *Trollinger*, *Bielak*, and *Deshler*, the theory that the pneumococcal vaccine “can cause” GBS seeks to demonstrate a link between indirectly-present components³⁴ of the vaccine and molecules found in myelin structures (in this case, lipids), but without additional sufficient evidence persuasively establishing likely pathogenicity. In effect, claimants seek application of the flu vaccine-GBS association in a context in which it does not work—and to accept it would be to find causation mostly because a claimant’s GBS post-dated vaccination, reverse-engineering causation from the temporal association.

II. Petitioner Has not Carried His *Althen* Prong One Burden of Proof

My analysis herein focuses solely on whether the pneumococcal vaccine “can cause” GBS.³⁵ As noted above, I have now on three occasions found that *this is unlikely*—and no new or alternative evidence was presented in this case that would provide compelling grounds for revisiting the issue, or finding the burden met here where it was not before.

In short, Dr. Steinman’s theory is unreliable and unpersuasive when evaluated in its totality (even if it does possess subcomponents that are scientifically reliable or reasonable in isolation). As in many cases, he embraces molecular mimicry as a mechanism³⁶ for how the vaccine’s components could cause the immune system to produce autoantibodies that would then attack myelin-associated structures that mimic vaccine antigens or components like the conjugate (which is not included in the vaccine for the purpose of encouraging immunity to it in the future specifically. But although molecular mimicry is well-established in the Vaccine Program as providing a reliable scientific explanation for how GBS may *often* occur after receipt of the flu vaccine specifically, it cannot be invoked summarily in all circumstances and then deemed to resolve causation. *McKown v. Sec’y of Health & Hum. Servs.*, No. 15-1451V, 2019 WL 4072113, at *50 (Fed. Cl. Spec. Mstr. July 15, 2019) (“merely chanting the magic words ‘molecular mimicry’ in a Vaccine Act case does not render a causation theory scientifically reliable, absent *additional evidence* specifically tying the mechanism to the injury and/or vaccine in question”) (emphasis in original), *mot. for review denied*, 76 Fed. Cl. 452 (2007)).

³⁴ By indirect, I mean only that the vaccine is not manufactured or engineered *to specifically include* phospholipids or phosphoglycerol—although the polysaccharide antigens in the vaccine *themselves* happen to contain these sub-structures, and their existence *within* the polysaccharides might have some internal significance.

³⁵ I thus do not also decide if Petitioner likely *had* GBS, or if he met the other two *Althen* prongs. The question of diagnosis is in fact hotly-disputed, and would have to be addressed were my *Althen* prong one finding deemed in error. However, because of my prior exposure to the questions surrounding this aspect of causation, I find it in the interest of judicial efficiency to evaluate only the “can cause” question herein.

³⁶ Although Program claimants are not *required* to propose a mechanism in support of a causation theory, they often attempt to do so—and thus invite reasonable scrutiny into whether the proposed mechanism is persuasive and/or has preponderant support. *Andreu*, 569 F.3d at 1378–79 (citing *Capizzano*, 440 F.3d at 1325–26).

Here, there are too many foundational deficiencies and holes in the overall theory to embrace molecular mimicry as likely driving GBS after receipt of the pneumococcal vaccine. At the outset, Dr. Steinman cannot explain convincingly why the pneumococcal vaccine would be more likely to cause GBS than its wild bacterial infectious analog (which unquestionably is *not* so associated). He vaguely points to the conjugate as the “X factor,” but his theory does not rely on it as explanatory (and in fact proposes a separate mechanism involving the conjugate that could independently also cause GBS, although he admits this is a less-substantiated theory (*see* Tr. at 6)). In fact, Dr. Whitton persuasively explained (offering a comparison of *C. jejuni* with *S. pneumoniae*) why one bacterium is known to be associated with GBS, while the other is not, noting that this likely has something to do with the differing polysaccharide capsules that protect the bacteria. First Whitton Rep. at 5. Although it is not a baseline requirement in Program cases to prove a vaccine’s wild infectious counterpart is associated with a particular disease, claimants certainly highlight the connection when it exists—and it is absent herein.

Next, Dr. Steinman proposes an antigenic/autoimmune self-target for GBS’s pathogenesis—polar head groups containing phospholipid components—that is not accepted overall by medical science specific to GBS, let alone addressed in reputable publications or studies. (In effect, if Dr. Steinman is correct, he has made a major discovery about GBS that others in the field more well-versed in the specific study of peripheral neuropathies *have fully missed*. Compare Hughes, Kwan). It is simply speculative to propose that GBS could be *mediated by an attack on this target*, based solely on the logic that the myelin *contains* it—and the degree of speculation is highlighted by the comparatively larger amount of evidence that associate gangliosides as a likely target. He further assumes the vaccine can cause antibodies specific to phospholipid structures to be produced when that is not even the primary intent of the vaccine, and relies for support on case reports or literature that expressly acknowledges that the *presence* of these antibodies in the blood sera of GBS patients does not mean they were *causal* of GBS. *See, e.g.,* Gilburd, Nakos. And he also cannot show that the anti-phosphoglycerol autoantibodies would react against the gangliosides either.

Many items of evidence offered in support of Dr. Steinman’s theory may have individual reliability, but do not appreciably aid construction of his theory. He repeatedly, for example, invokes literature specific to MS—not the disease at issue, even if it has some common features with other demyelinating conditions. (The same goes double for his citation to recent, admittedly ground-breaking research about an association between MS and the EBV infection). He identifies studies in which the presence of anti-phosphoglycerol antibodies are seen in the blood sera of GBS patients, but where the study’s authors do not propose to explain the role of the autoantibodies. Chang at 7091–92; Gilburd at 27. And he goes to great lengths to attempt to argue that phosphoglycerols *must* be in the pneumococcal vaccine, or play some role in its immunogenicity—when that only “sets the stage” for his mechanistic arguments that fail to persuade for many other reasons.

Dr. Steinman’s secondary causal theory—that the conjugate in the vaccine is an alternative source for a protein (rather than sugar) molecular mimic comparable to a myelin component—fares no better. Indeed, although this aspect of the theory was not at issue in *Trollinger* or *Bielak*, it was in *Deshler*, and Dr. Whitton (who served as Respondent’s expert in both that case and this one) has now effectively rebutted the concept twice. As I noted in *Deshler*, CRM₁₉₇ is lab-created, and although it is *comparable* to diphtheria toxin, it is not the same, reducing the value of analogizing the results of research articles like Raju to the present context. *Deshler*, 2020 WL 4593162, at *11. In addition, while the conjugate’s inclusion in the pneumococcal vaccine is intended to boost immunogenicity, the conjugate is not *itself* added to provide an additional beneficial antigenic component (since Prevnar-13 aims to protect against pneumococcus bacteria—not diphtheria), further reducing the likelihood that it would cause the production of antibodies specific to it that would in turn cross-react with self mimics. Dr. Steinman’s speculative theory about the immune response to this ingredient is otherwise unsupported by sufficient science specific to the conjugate itself and its purported capacity to induce an aberrant response. And Petitioner offered hardly any evidence (in comparison to the other aspect of his theory) that vaccine recipients mount a response to the conjugate that would *produce* Contactin-1 antibodies, let alone that Contactin-1 is a likely GBS pathogenic target.

Other arguments for causation were no more persuasive, although of lesser significance. For example, although both experts spent a great deal of energy arguing about the utility of BLAST searches as well as Dr. Steinman’s preferred analytical structure for evaluating causation, this case does not turn on whether a five, ten, or twenty amino acid sequence must be shown to establish reliable homology. Rather, as I have noted many times now in prior decisions (often in response to Dr. Steinman’s “in silico” desktop database search efforts), *more* than homology must be shown to preponderantly demonstrate that a vaccine likely causes cross-reactive damage via the mechanism of molecular mimicry. *See, e.g., Trollinger*, 2023 WL 2521912, at *29; *Schilling v. Sec’y of Health & Hum. Servs.*, No. 16-527V, 2022 WL 1101597, at *5, 19–20 (Fed. Cl. Spec. Mstr. Mar. 17, 2022); *McKown*, 2019 WL 4072113, at *50. Dr. Steinman’s success in establishing sequential homology (ignoring Dr. Whitton’s reasoned objections to the utility of BLAST searches) does not make it more likely the pneumococcal vaccine causes GBS without other corroborative evidence that has not been offered in this case. Certainly nothing new and on point was filed to that end that I have not already considered in my prior cases involving the pneumococcal vaccine.³⁷

³⁷ Lanz or Robinson & Steinman do qualify as more recent publications, but *they involve MS and a different viral infection*, not GBS and pneumococcus bacteria. Accordingly, the fact that they might provide ballast for Dr. Steinman’s argument about what amino acid chain length is sufficient for a cross-reaction is of little regard. Indeed, the existence of such studies—which have been hailed as breakthroughs in proposing a possible infectious trigger for MS—underscore the comparative lack of comparable reliable proof *in this case*. Where is the study showing a similar link between GBS and *S. pneumoniae*? The usual excuses offered for why more specific evidence was not possible (it cannot be done; the disease is too rare; it is unfair to demand such proof in a field bereft of certainty; etc.) wilt in the face of strong research supporting *other* causal connections in disparate contexts.

I similarly do not give great weight to the fact that some treaters speculated that the vaccine was causal of Petitioner's GBS. I acknowledge that such evidence has relevance to the *Althen* prong one inquiry, even though treater views usually are thought to have more significance in the context of determining whether the second, "did cause" prong has been satisfied. But it is not evident from the record that treater speculation on this subject was informed by the scientific specificity displayed by both sides' experts on prong one. And the case report evidence (a class of proof not usually deemed deserving of significant weight as it is) offered herein, like El Khatib, was especially thin, and hardly supported Dr. Steinman's more sweeping contention that a GBS-pneumococcal vaccine association is "recognized." Second Steinman Rep. at 2.

Petitioner endeavored to fend off the weaknesses in his theory, but was unsuccessful. For example, he argues that gangliosides are not necessarily the *exclusive* target in GBS (and that in any event, it cannot be shown in this case that he possessed antibodies to them). This, however, misapprehends the importance of the target "question." Dr. Whitton established that medical science already knows a great deal about *likely* GBS targets, comparing that information to the fact that GBS authorities generally have *not* embraced phospholipid polar group heads as a target (or *S. pneumoniae* as a trigger for that matter). And numerous items filed in this case *specific* to GBS note that no conclusions can credibly be drawn about the role anti-phosphoglycerol antibodies play in GBS, even if they are found in the blood of GBS patients. Nakos at 1406–07; Gilburd at 23. It is also incorrect for Petitioner to suggest gangliosides are only relevant when GBS is thought to have been caused by a *C. jejuni* infection, since in numerous Program cases gangliosides as a target are discussed in the context of *vaccine* causation. *Pierson*, 2022 WL 322836, at *24 (remarks from another special master that note "[f]lu vaccine-GBS cases often rely upon the theory of molecular mimicry, specifically proposing that antibodies produced by B cells in response to the vaccine's viral antigen components cross-attack the myelin sheath (where the target antigen and gangliosides of the myelin sheath share structural homology), causing demyelination of peripheral nerves.")

Respondent, by contrast, effectively and persuasively rebutted Petitioner's theory. Dr. Whitton could not deny that phosphoglycerol is found in at least some of the vaccine's 13 polysaccharide antigens. But he established (based on the literature filed herein) that it is unlikely that (a) antibodies *are* generated to the phosphoglycerol-specific aspects of some of the vaccine's antigenic serotypes, (b) they can *in turn* react with host tissue phospholipid polar head structures, or (c) GBS is *primarily or initially* mediated by such a cross-reaction, with articles like Nakos and Gilburd admitting that the existence of such antibodies (which could be the *product* of an ongoing disease started by something distinguishable) does not in turn imply pathogenic primacy. Indeed, as other Program cases have acknowledged, antibodies often exist or are found in blood sera but are not definitionally pathogenic. *Scott v. Sec'y of Health & Hum. Servs.*, No. 03-2211V, 2006 WL 2559776, at *19 (Fed. Cl. Spec. Mstr. Aug. 21, 2006) ("Some agents may trigger the production of antibodies without also triggering the disease."). I give more weight overall to Dr. Whitton when opining on the biochemistry and immunologic topics relevant to the causation theory.

I do not find *all* of Petitioner’s arguments unpersuasive or (at least in this narrow context) unreliable. For example, this is not a case in which persuasive epidemiologic evidence weighs against the offered causation opinion. Articles like Haber, Tseng, and Baxter either involve the kind of passive data never deemed particularly compelling in Program cases, or did not involve reliable findings based on a comparison of results specific to a vaccinated sample against an incidence rate for the unvaccinated. My prior decisions consistent with the present, like *Bielak* and *Trollinger*, also did not turn on the weight given to such items of evidence. (Of course, I do not find persuasive Petitioner’s suggestion that these studies or articles establish a medical community consensus in favor of a pneumococcal vaccine-GBS relationship—if anything, *the opposite is true*, as Dr. Whitton proposed, when he pointed out how existing GBS literature never proposes that the wild *S. pneumoniae* infection is associated with GBS. But either way, the existing epidemiologic studies discussed in this case by both sides were not probative of a relationship or lack thereof).

In addition, reliable evidence establishes that individuals with central nervous system neuropathies, like MS, have been shown in small sample studies to possess antibodies specific to myelin-containing phospholipids. I do not find reason to dispute the homologies observed in Dr. Steinman’s BLAST searches, or that, despite Dr. Whitton’s contentions, the chain lengths were inadequate (although as already noted the case turns on more fundamental questions of GBS pathogenesis than homology).³⁸ And Petitioner did show that phosphoglycerols are found in both *some* of the vaccine’s antigens as well as nerve myelin.

But it remains *preponderantly unlikely* that anti-phospholipid/phosphoglycerol antibodies drive GBS in its initial stages, or that they do so by targeting polar group heads specifically, or that the pneumococcal vaccine causes GBS in a way that other medical science has yet to recognize. What remains is a theory that does no more than attempt to “connect the dots” between vaccine components and some molecular components of nerve myelin—a theory that largely hopes to replicate the logic supporting a link between the flu vaccine and GBS. But that vaccine is not interchangeable with the pneumococcal vaccine. They have wholly different ingredients and function differently, and are also aimed at different kinds of illnesses.

III. Petitioner Misapprehends the Evidentiary Weighing Process

As noted, the parties opted for oral argument by counsel in lieu of a hearing. This afforded Petitioner’s counsel not only the chance to highlight the aspects of his case he deemed most

³⁸ I thus do not give great weight to Dr. Whitton’s argument that Dr. Steinman’s practice of employing BLAST searches constitutes a misuse of the database, and I do not dispute the baseline utility it provides him in establishing potential homologic sequences when comparing a variety of compounds, protein or otherwise, common to vaccines and human tissue—although the science-heavy showing he made on this front did more to obscure and confuse than support the theory that the vaccine can cause GBS.

important, but also to comment on *how* his prong one showing should be evaluated, in light of the Vaccine Program’s preponderance evidentiary standard.

At argument, Petitioner proposed that I might be mistakenly “raising” his burden in the context of the evidentiary weighing that special masters perform. He maintained that the evidence offered was sufficient to meet his burden, since Respondent could not show that the scientific literature was unreliable or false, and Dr. Steinman was qualified to opine on the subject. In addition, the fact that other special masters had found this same showing adequate highlighted my error. These arguments merit comment.

There is no doubt that the Program is structured in many ways to be favorable to its claimants to serve the policy goal of encouraging vaccination—in particular, by ensuring a means of compensating those injured by a vaccine through no fault of their own. No rules of evidence, for example, limit the proof that can be offered and considered; on the contrary, an expansive array of evidence, more often than not circumstantial, is reasonably relied upon and evaluated when deciding if the *Althen* prongs are met. In addition, cases are supposed to be resolved expeditiously,³⁹ with the special masters applying “inquisitorial” methods to direct a case toward its proper conclusion rather than passively conducting proceedings. And to ensure a competent bar exists to represent injured parties, the Act incorporates perhaps the most generous fee-shifting provisions found in the federal system, paying fees in failed cases as often as in successful matters.

All of the above, however, does not mean that petitioners prevail simply by offering evidence in an exercise of “checking off” boxes from the *Althen* framework, then stepping back and declaring their work is done. Rather, the balance of evidence is necessarily *weighed* by the special master—not only in an initial, prima facie sense (for some cases do not even cross that threshold), to determine that *some* evidence relevant to each prong has been offered, but also to determine how Petitioner’s arguments and proof stack up against what Respondent marshals. For a finding of entitlement, the probative value/quality and persuasiveness of a petitioner’s evidence, considered *in toto*, must at least *barely* outweigh the Respondent’s. *Some* overall mix of evidence must exist suggesting that a given vaccine could cause an injury (and, where a mechanism is proposed, that this mechanism was preponderantly established).

After repeated careful consideration of the science and medical thinking about the pneumococcal vaccine and GBS, I have concluded that the proof available is simply too insubstantial to preponderantly show the pneumococcal vaccine can cause GBS. Petitioner is only able to identify some molecular consistency between sub-components of the vaccine and myelin,

³⁹ This goal has been hard to meet in the past ten-plus years, owing to the ever-growing number of cases filed in the Vaccine Program—but it remains a very important consideration, and one I am always mindful of when processing claims and scheduling cases for trial or other resolution.

coupled with other evidence suggesting (from small test samples, moreover) that antibodies relevant to these consistent components might *exist in the context of GBS*. This does not mean that it is *likely* they drive GBS (especially given how much is known about the exogenous factors that likely *do* drive GBS, or the self antigenic targets), or that they do so by attacking myelin structures wholly distinguishable from those already believed by science to be autoimmune targets.

Is my determination simply masking a demand for scientific certainty? Only if the weighing process I am tasked with performing did not involve consideration of Respondent's arguments and evidence, or if it were limited solely to evaluation of the reliability of individual scientific studies or the credentials and expertise of a claimant's expert—as Petitioner suggested in oral argument. But *this is not how special masters function*, as the Court has recognized. On the contrary—I am properly tasked with weighing both the probative value of individual items of evidence and the credibility and persuasiveness of an expert's testimony against the evidence offered *contra*. I am never compelled to accept what is offered at face value—by either side—any more than any fact finder would be. *Sword v. United States*, 44 Fed. Cl. 183, 188 (1999) (“[e]ven more than ordinary fact-finders, this Court has recognized the unique ability of Special Masters to adjudge cases in the light of their own acquired specialized knowledge and expertise . . . The Special Master's sole professional responsibility for years has been to preside over vaccine cases . . . No judge or jury can be forced to accept or reject an expert's opinion or a party's theory at face value”). Denying entitlement after performing such weighing, in a quest for sufficient evidence, does not mean certainty was demanded. *Hodges v. Sec'y of Health & Hum. Servs.*, 9 F.3d 958, 962 (Fed. Cir. 1993) (noting rejection of medical opinion on causation did not amount to requiring certainty, but instead reflected the special master's inquiry into whether “some degree of acceptable scientific support” existed to conclude preponderance standard had been met).

If anything, Petitioner's arguments reflect a desire to *lower* his burden—something the Circuit instructs is as inappropriate as requiring certainty. *Boatmon*, 941 F.3d at 1359–60. But in some circumstances, the existing scientific and medical evidence relevant to a particular disease and vaccine simply does not support causation, and it is no objection to maintain in response that what a petitioner *can* offer should be deemed sufficient if it is “science-y” enough. *Caves v. Sec'y of Health & Hum. Servs.*, 100 Fed. Cl. 119, 143 (2011), *aff'd*, 463 F. App'x 932 (Fed. Cir. 2012) (“the standard of proof does not operate as a sliding scale that varies depending on the quantity and quality of the scientific evidence that is available”). It has not been shown to be likely that the pneumococcal vaccine can cause GBS, because the evidence that exists on the topic is too sparse overall to constitute a persuasive preponderant showing, after the weighing of evidence pro and con has been performed.

Finally, what of the fact that other special masters have reached a different result? In many cases, because the mix of evidence and nature of the theory presented from case to case can vary, inconsistent outcomes are easier to accept. Not so here. And while special masters often take refuge in the legal truism that their colleagues' determinations do not bind them, Petitioner not-unreasonably notes the fact that I am (so far) alone in my negative assessment of this causation theory.

In response, I return to my analysis herein (which recapitulates *Bielak* and *Trollinger*) highlighting the specific weaknesses of the theory that GBS can be caused by the pneumococcal vaccine. As I have now exhaustively stated several times, the evidence associating the vaccine (along with its wild bacterial cognate) with GBS is notably weak—substantially weaker than the evidence linking the flu vaccine, or even wild flu virus. Arguments that antibodies produced in response to the pneumococcal vaccine can attack nerve structures and instigate GBS rely on articles I discussed at length in *Bielak* and *Trollinger*—but found wanting. *See Bielak*, 2022 WL 18058244, at *15–17, 32 (discussing Chang, Gilburd, Ho, Kanter, and Nakos); *Trollinger*, 2023 WL 2521912, at *28–29 (same). Those same articles were offered in the successful cases. *See, e.g., Gross*, 2022 WL 9669651, at *15–17, 28; *Pierson*, 2022 WL 322836, at *12, 16. And Dr. Whitton convincingly explained why it could not be assumed, and had not been shown, that an antibody produced in response to one of the vaccine's phosphoglycerol-containing polysaccharides would even be recognized by a myelin phospholipid structure, let alone attack the polar head groups (which have not been shown to be likely GBS targets in any event). A bare demonstration of a cross-reactive *potential* is not enough to prevail, especially in the absence of corroborative evidence that associates GBS with the pneumococcal vaccine or its wild bacterial counterpart. *Bielak*, 2022 WL 18058244, at *33, 36. And I have also previously addressed comparable arguments about the role of the conjugate. *Deshler*, 2020 WL 4593162, at *20–21.⁴⁰

Overall, the opinion offered herein is wholly consistent with theories I have reviewed—and rejected—three times before. My prior, and present, determinations were the product of careful evaluation of the evidence, expert reports, and scientific literature (with identical items filed in all of these matters). I did not accept at face value Petitioner's arguments for what the literature says, but read the filed items carefully, weighing them against Respondent's criticisms. Although I would always prefer to be guided by the collective wisdom of the other special masters, I do not deem the cited counter-determinations to be sufficiently persuasive in their analysis to suggest that I am in error. Rather, like *Boatmon*, those cases may reflect instances in

⁴⁰ In fact, although my determinations in *Trollinger* and *Bielak* squarely contend with the findings in some other decisions, my determination in *Deshler* with respect to the conjugate was not addressed in such cases, which distinguished it on the grounds that Dr. Steinman's phosphoglycerol theory had not been offered in *Deshler*. *See, e.g., Gross*, 2022 WL 9669651, at *36; *Pierson*, 2022 WL 322836, at *26, 31.

which the proper evidentiary standard was inadvertently lowered, based on the unexamined assumption that what “works” for the flu vaccine-GBS association works for *any* vaccine.⁴¹

CONCLUSION

Claimants must carry their burden of proof—here, by preponderantly establishing, via an offering of sufficient evidence *specific to the pneumococcal vaccine in question*, how it could cause GBS. This has not been accomplished in this case. Accordingly, I deny entitlement.

In the absence of a motion for review filed pursuant to RCFC Appendix B, the Clerk of the Court **SHALL ENTER JUDGMENT** in accordance with the terms of this Decision.⁴²

IT IS SO ORDERED.

/s/ Brian H. Corcoran
Brian H. Corcoran
Chief Special Master

⁴¹ Of course, if (as was determined in prior cases) it is enough to find that a vaccine has *some* cross-reactive potentiality with self tissues on the basis of Dr. Steinman’s argument, it may as well be concluded that *all* covered Program vaccines are capable of causing GBS, so long as an expert like Dr. Steinman is willing to provide a series of lengthy expert reports so opining. At that point, of course, the special masters would be creating a new Table claim.

⁴² Pursuant to Vaccine Rule 11(a), the parties may expedite entry of judgment if (jointly or separately) they file notices renouncing their right to seek review.